THE
AMERICAN
SURGEON

Vol. 23, No. 6 June 1957

CARDIAC STAB WOUNDS

CHAMP LYONS, M.D., REX PERKINS, M.D.

Birmingham

For many centuries wounds of the heart were viewed with fatalistic mysticism as being beyond hope of salvage, a viewpoint augmented by the ancient teaching of Hippocrates,[32] Aristotle,[2] and Galen.[29] This viewpoint was challenged in the seventeenth century by the onset of an era of observation and experiment.[3] It began to be appreciated that a cardiac wound may not necessarily be fatal,[56] and isolated cases of survival with healing were reported, culminating in Fischer's[28] compilation in 1868 of 452 cases of which 10 per cent of the patients recovered. The effects of cardiac compression were first mentioned by Morgagni[33, 44] in 1761 and in 1884 Rose[52] invented the expression *"heart tamponade"*, and presented cases of death due purely to this syndrome. The last 2 decades of the nineteenth century produced considerable experimental work[12, 19, 25, 53] on the production, suture, and healing of cardiac wounds, first accomplished by Block[11] in 1882. This work met with the expected resistance, culminating perhaps in the famous statement by Paget[47] in 1896—that "surgery of the heart has probably reached the limits set by Nature to all surgery: no new method and no new discovery can overcome the natural difficulties that attend a wound of the heart. It is true that *'heart suture'* has been done on animals, but I cannot find that it has ever been attempted in practice." Actually the first case of suture of the human heart had been done in 1895 by Cappelen,[14] the patient dying 2½ days later. In 1896 Farina's[26] patient lived 8 days, dying of bronchopneumonia. In 1897 Rehn[51] sutured an actively bleeding 1.5 cm. wound of the right ventricle with recovery of the patient. Many successes followed, and in 1920 Tuffier[60] collected 305 cases with a recovery rate of 50.4 per cent. The first successful suture of the human heart in this country was done by Dr. Luther Hill,[31] of Montgomery, Alabama, on Sept. 15, 1902. The patient, a 13 year old Negro boy, had been stabbed the previous afternoon, developed tamponade, and was operated upon at 1 a.m. in a Negro cabin by the light of a kerosene lamp. He was in good health 40 years later.

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

Surgical cardiorrhaphy became the accepted treatment, abetted by increasing knowledge of surgical technic[3] and advances in anesthesia and blood replacement. Meanwhile experimental work continued on the production and treatment of acute pericardial tamponade.[3, 15, 36] It was found in the dog that an acute pericardial pressure of 12 to 22 cm. H_2O or a volume of about 150 cc. produced death[3, 17] and that the animal could be easily salvaged by reducing the pressure by removing only a fraction of the fluid. A rapid intravenous infusion enabled the animals to withstand up to 146 per cent greater pressure.[17] These experimental findings were applied to the therapy of human patients in the form of pericardial tap and intravenous infusion of fluid and blood, with the result that selected cases of acute tamponade were successfully managed nonsurgically. Meanwhile there was increasing concern over the continuing high mortality rate associated with operative cardiorrhaphy (fig. 1), ranging from 25 to 50 per cent. Thus about the time of World War II began the era of increasing emphasis on the conservative management of cardiac stab wounds in which the presenting clinical picture was that of acute pericardial tamponade. It has been convincingly demonstrated by Blalock, Elkin, Bigger, and others that this method of management, in selected cases, is sound and is attended by very low mortality (fig. 2). Certain more recent contributions to the pathophysiology of

Series of statistics on pentrating wounds of the heart treated by thoracotomy

Reporter	Year	Cases	% Mortality
Peck[48]	1909	160	63
Pool[49]	1912	77	45.5
Smith[58]	1923	49	28.6
Schoenfeld[55]	1926	25	36
Bigger[4]	1932	53	26.5
Bigger[5]	1939	141	50
Elkin[21, 22, 24], Griswold[30], Nelson[46], Linder[37], Blau[10]	1941 to 1945	149	38

FIG. 1

Series of statistics on penetrating wounds of the heart treated by pericardial tap

Reporter	Year	Cases	Deaths
Bigger[6]	1940	3	0
Blalock[9]	1943	2	0
Blau[10]	1945	3	0
Anderson[1]	1946	4	0
Ravitch[50]	1949	9	1
Elkin[24]	1951	11	0
Menendez[42]	1951	4	1

FIG. 2

tamponade include the demonstration[54] that ventricular diastolic filling, and thus cardiac output, is impaired; and the experimental finding[13] that in dogs subjected to a standard heart wound, 80 per cent of deaths occur in the first 30 minutes, implying that patients surviving long enough to reach the hospital are in a select group more likely to survive. In most clinics today the prevailing attitude seems to be that initial therapy of penetrating heart wounds with tamponade should be pericardial aspiration, repeated if necessary, and that surgery is performed if tamponade continues to recur.

In Birmingham, we have maintained an interest in the primary operative management of these cases. During the past 4 years, 14 additional patients have been operated upon, with 2 deaths (chart I, fig. 3). Several points of interest are seen in these patients. All but 3 of the patients were intoxicated. All but 1 were in shock, and in 9 there was no blood pressure on admission. In 4 patients the problem was exsanguination, while in 10 it was primarily tamponade as demonstrated at thoracotomy. In 5 patients pericardial tap was done with interesting results: In patient number 1, tap yielded 40 cc. of blood, with some improvement of blood pressure, but at surgery there was a marked tamponade. In patient number 2, tap was negative, but at surgery there was tamponade consisting entirely of clot. In patient number 5, the tap yielded 140 cc. of blood with no clinical improvement. In patient number 6, the first tap yielded 100 cc. of blood with return of pressure to 80/40, a later tap yielded nothing and at surgery there was tamponade. In patient number 9, tap was unproductive and there was a tamponade. The precarious state of many of these patients on admission is indicated by the finding that in 2 patients thoracotomy was necessary in the emergency room and in 1 patient en route to the operating room. In 5 patients cardiac resuscitation was necessary, with 2 deaths. One of the 2 patients who had ventricular fibrillation survived. In 2 patients a major coronary vessel was severed, 1 surviving. In 11 patients the pericardial content was primarily clotted blood. In all patients the cardiac chamber was penetrated, in 10 the wound was actively bleeding, and in the other 4 it reopened prior to suture. In the survivors the average hospital stay was 15 days, and there were no complications except the patient who was blind. In patient number 7, who died of an automobile accident 5 months later, autopsy showed mild adhesive pericarditis without constriction, and a healed myocardium without endocardial change. It was noted in all patients that induction of general anesthesia caused further cardiovascular collapse and loss of what blood pressure was present. Thus the surgery should be directed toward rapid, calm, and deliberate operative relief of the tamponade and control of the bleeding.

In view of the preponderance of material in the recent literature emphasizing the nonoperative approach to cardiac stab wounds, it might be in order to briefly mention some considerations which throw a more favorable light upon a policy of treatment by thoracotomy for many of these patients.

1. The surgical mortality in certain well controlled series has been quite low. Griswold,[39] for the years 1938 to 1947 reported 36 cases with 3 deaths. Jordan[34] reported 24 cases with 3 deaths. It must be remembered that in many instances

CHART I

Case	Date Operated	Age	Sex	Race
1. R. K.	11–9–52	31	M	C
2. J. C.	9–27–53	34	M	W
3. M. M.	11–24–53	33	M	C
4. J. M.	11–26–53	38	M	C
5. H. D.	2–27–54	24	M	C
6. J. C.	9–25–54	39	M	C
7. G. P.	2–9–55	35	F	C
8. L. G.	2–27–55	34	M	C
9. A. H.	6–12–55	46	M	C
10. W. E.	3–17–56	19	M	C
11. J. D.	7–1–56	24	M	C
12. J. M.	7–7–56	60	M	C
13. T. J.	7–14–56	27	M	C
14. J. W.	7–26–56	19	M	C

	Tamponade				
General condition	Present	BP	P	VP	X-ray
1. Comatose, profound shock.	No	0	0	128	No
2. Admitted 11 hrs. after injury.	Yes	90/60	0	Elevated	Left hemothorax. Fluo —good pulsation.
3. Pale. Cold. Resp. distress.	Yes	80/60	?	350	No
4. Comatose. Nonresponsive. No heart sounds.	Yes	0	0	Not done	No
5. Unconscious. Heart Sounds inaudible.	Yes	0	Weak	Not done	No
6. Comatose.	Yes	0	0	340	Sl. enlarged heart shadow.
7. Shock. Ten deep lacerations of chest, neck, upper extremities.	Yes	0	Weak	225	Pneumohemothorax, left. Enlarged heart shadow.
8. Shock.	Yes	0	Weak	Not done	No
9. Shock.	Yes	70/0	90	Not done	No
10. Comatose. Shock.	Yes	0	0	Not done	No
11. Shock.	No	0	140	Not done	No
12. Unconscious. Shock. No heart sounds.	Yes	0	Weak at 60	100, 160	Large heart shadow.
13. Stuporous.	No	60/30	Rapid Weak	140	Left hemothorax. Mediastinal shift to right.
14. Shock.	No	70/50	70, weak	Not done	Left hemothorax.

CHART I.—*Continued*

Surgery				
Pre-op Treatment	Major Findings	Pericardial Contents	Site of hole	Hole closed or bleeding
1. Transfusion left thoracentesis yielded 500 cc. 15 min after admission resp. ceased. Thoracotomy in Emer. Rm. Cardiac massage. Carried to O.R. with BP 60/0.	0.5 cm hole in main PA. 1000 blood left chest	"Small amount" blood	Main PA	Closed but reopened on manipulation
2. Pericardial tap neg.	1 cm hole in main PA Moderate left hemothorax.	150 cc. clot	Main PA	Closed but reopened on manipulation
3. Pericardial tap yielded 40 cc. blood c̄ BP rise to 90/60.	Tense, blue pericardium 2 cm. hole in RA	"About 500 cc. blood" "Large amount" blood and clots	RA Thru & Thru of RA	Bleeding
4. None. Thoracotomy performed in Emer. Rm. on admission. Cardiac massage. Massive transfusion.	"Tremendous tamponade" No heart action. After 1 min. massage, heart action resumed. Rt. coronary artery lacerated & bleeding.			
5. Pericardial tap yielded 140 cc. blood s̄ improvement.	5 mm. hole in RV	300 cc. blood & clot	RV	Bleeding
6. Pericardial tap yielded 100 cc. with return to BP to 80/40 pulse 102. After 2 hrs. repeat tap yielded nothing. Lt. thoracentesis yielded 80 cc. blood BP remained at 80–90 systolic. Surgery 4 hrs after adm.	Distended pericardium 1 cm laceration RV	200 cc. clot with some blood	RV	Closed, but reopened on on manipulation
7. 2500 blood. Bilateral thoracentesis neg. Abdominal tap neg. Tube inserted into left chest. Repeat lt. thoracentesis in OR yielded 600 cc. blood.	3 cm ragged hole in RV. Distended pericardium. Large left hemothorax.	200 cc. clot and blood	RV	Bleeding

CHART I.—*Continued*

Surgery				
Pre-op Treatment	Major Findings	Pericardial Contents	Site of hole	Hole closed or bleeding
8. 1000 blood. Pressure rose to 100, then quickly fell to 0 again. Left thoracentesis yielded 350 blood.	Small amount blood in left chest. 1 cm. laceration RV.	"Moderate amount" blood & clot	RV	Bleeding
9. Left thoracentesis. Pericardial tap yielded "small amount" blood, no elevation of BP resulted.	1 cm. laceration RV. Small amount blood in left chest.	300 clot with some blood	RV	Bleeding
10. Taken directly to OR. Resp. ceased en route, and thoracotomy done on stretcher. 2000 cc. blood.	Ventricular fibrillation. Distended pericardium. Lt hemothorax. 1 cm. laceration LV & ant. desc. artery.	"Large Amount" blood	LV	Bleeding
11. Thoracentesis negative. 500 cc. blood brought BP to 100/70.	1.5 cm laceration RV. 800 cc. clotted blood in left chest.	"Moderate Amount" clot	RV	Closed, but reopened on manipulation
12. 500 blood brought BP to 70/0, quickly dropped to 0 again	1 cm. laceration LV 900 blood in lt chest.	500 cc. clot and blood	LV	Closed
13. Transfused. BP remained at 60.	2 cm. laceration LV Large left hemothorax.	"Large Amount" blood & clot	LV	Bleeding
14. Transfused. BP remained at 70. Left thoracentesis yielded 1500 cc. blood.	1 cm. laceration RV. Large left hemothorax.	"Large Amount" blood & clot	RV	Bleeding

CHART I.—*Continued*

Surgery				
Procedure	Complications	Result	Comment	Follow-up
1. Suture of hole. Tracheotomy.	None	Survived		Well 2-3-55
2. Suture of hole.	None	Survived		Well 10-20-56
3. Suture of hole.	None	Survived		Well 4-8-54
4. Suture of holes. Lacerated coronary, ligated. Tracheotomy Cardiac massage.	EKG showed auricular fibrillation, myocardial ischemia. Patient awakened, but remained in shock despite 3000 cc. blood. Incontinent. Anuric.	Died 10 hours postop.	At end of surgery BP was 110/70, but quickly slipped to 70/50 with pulse 50. This death seems due to coronary artery damage.	
5. Suture of hole.	None	Survived		Well 10-6-54
6. Suture of hole.	None. Postop venous pressure 120.	Survived		Well 11-26-54
7. Suture of lac. not successful. Ant. desc. left coronary sutured with acute infarct. Cardiac massage. Defibrillation unsuccessful.	Friable myocardium would not hold sutures. Ventricular fibrillation. 6500 cc. blood given, total.	Died on table 8 hrs. after admission	After initial transfusinos, BP rose to 110/80, quickly slipped to 0 again. Should have had pericardial tap. Death due to exsanguination plus acute surgical infarct.	
8. Suture of hole.	None	Survived	Should have had pericardial tap.	Died 7-28-55 of cerebral trauma after auto accident. Post showed chronic adhesive pericarditis. No RV thrombi.
9. Suture of hole.	None	Survived	Pericardial tap failed to remove clot or alleviate tamponade.	Well on discharge. No follow-up.

CHART 1.—*Continued*

Procedure	Complications	Result	Comment	Follow-up
10. Cardiac massage. Defibrillation. Suture of hole. Ligation of coronary.	Myocardial infarct. Blindness.	Survived	Blindness due to cortical ischemia during arrest, or to methyl alcohol.	Well except blind, 10-20-56
11. Suture of hole.	None	Survived	Operated because heart could be palpated through chest wound.	Well 10-20-56
12. Suture of hole.	None	Survived		Well 10-20-56
13. Suture of hole. Cardiac massage.	None	Survived		Well 10-20-56
14. Suture of hole.	None	Survived		Well 10-20-56

the patients submitted to surgery will be the more seriously injured individuals who have the worst prognosis with any form of treatment.

2. Just as in abdominal wounds, it is desirable in thoracic wounds to have accurate information regarding the site and extent of internal injuries. The site of cutaneous puncture and the direction of a knife thrust are notoriously misleading. There may be injury of the aorta, esophagus, trachea, or lung. The type and length of the knife or other instrument is generally unreliable information even if it is known. Thoracotomy allows an accurate assessment of intrathoracic injury and a definitive management of the total pathology.

3. In regard to the heart itself, there is no way of knowing which chamber is injured, or whether an intrapericardial great vessel is injured. The symptoms of tamponade are the same regardless of the source of bleeding. It is believed[3, 13] that there is some prognostic difference involved depending upon the site of cardiac injury, the ventricles, aorta, and large coronary vessels giving rise to more serious bleeding, tamponade, and death. Hemorrhage from the proximal aorta can be controlled, and it is desirable to ligate or repair a severed coronary vessel.

4. The fate of a patient with a cardiac stab wound depends, among other things, upon the relative sizes of the myocardial and pericardial holes. With a small hole in each structure, some tamponade will probably develop, and this patient will likely respond well to tapping. With a small hole in the myocardium and a large hole in the pericardium, bleeding occurs into the pleura or externally, with some danger of exsanguination. There would be no indication for tapping. With a large hole in the heart and a small hole in the pericardium, acute severe tamponade would occur with a strong possibility of quick death. If this patient survived to be treated he would be likely to redevelop his tamponade after tapping and come to surgery. With a large hole in both myocardium and peri-

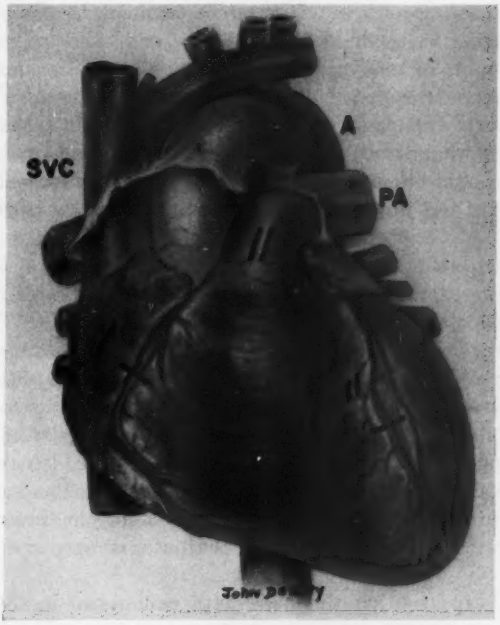


FIG. 3. Size and location of 14 cardiac stab wounds. The + signifies a through and through wound.

cardium, he would quickly exsanguinate, or if he survived to be treated, could be managed only by operation. There are certain other factors which affect the sealing of a cardiac wound, such as the thickness of the myocardium, the adherence of blood clot, and the displacement of the myocardial hole so it does not coincide with that of the pericardium. However, it can be seen that of the 4 situations described above, tapping will work in 1 patient, would be ineffectual in 2 patients, and of unpredictable efficacy in the fourth. This grouping of patients corresponds roughly to that promulgated by Bigger.[5]

Whereas a more logical basis for therapy could be reached if the patient's injury could be definitely placed into one of the above categories, we have not found that it is always possible to do so. These patients are usually of the lowest social order, usually intoxicated, uncooperative, and in shock. Venous pressure elevation may be delayed,[8] shows no rigid correlation to circulatory failure,[62] and is not easily performed accurately.

In many respects the findings in tamponade are similar to those of acute hemorrhage.[62] The information gained by pericardial or pleural taps may be inadequate due to the presence of clots. Roentgenologic examination involves delay in the definitive treatment of a critically ill patient and often yields equivocal information.[4] In many instances there are multiple thoracic injuries, or associated abdominal or head injuries which complicate the clinical picture.[41]

In our neighborhood, icepick injuries are not common, the usual weapon being the more damaging switch-blade knife. These and other considerations have caused us to doubt, in many cases, our own ability to accurately assess the extent of intrathoracic damage. We have successfully treated a number of patients by pericardial tap alone, but we have also been guilty, as others have[27, 50] of losing patients who arrived alive at the emergency room, and who died from 5 minutes to 6 hours later because we could not differentiate tamponade and exsanguination, or did not realize the type and extent of their intrathoracic injuries.

5. In addition to concern over lower mortality rate, some consideration should be given to lowered morbidity. Much of the intrapericardial blood will be clotted,[41] and blood clot cannot be removed through a needle. There is increasing evidence[20] that this clot gives rise to undesirable sequelae such as pericarditis, recurrent effusions,[61] and chronic constrictive pericarditis requiring decortication.[20] Experimentally, a healed cardiotomy wound gives a thicker and stronger scar if it is sutured than if it is not,[16] and it is probable that a number of cardiac wounds heal improperly. The initial clot may loosen, and certainly a number of cases are on record of delayed bleeding[40, 43] and recurrent hemopericardium with tamponade.[18, 57] Initial thoracotomy in these patients allows one to firmly suture the wound, and to evacuate, lavage, and drain the pericardium.

6. Pericardial tap itself has the disadvantages inherent in all such blind maneuvers and is potentially dangerous. In a recent questionnaire,[35] 18 out of 21 prominent cardiologists indicated personal knowledge of one or more deaths due to, or following upon, pericardial paracentesis. One doesn't know exactly where the point of his needle is and a negative tap may be merely an unsuccessful

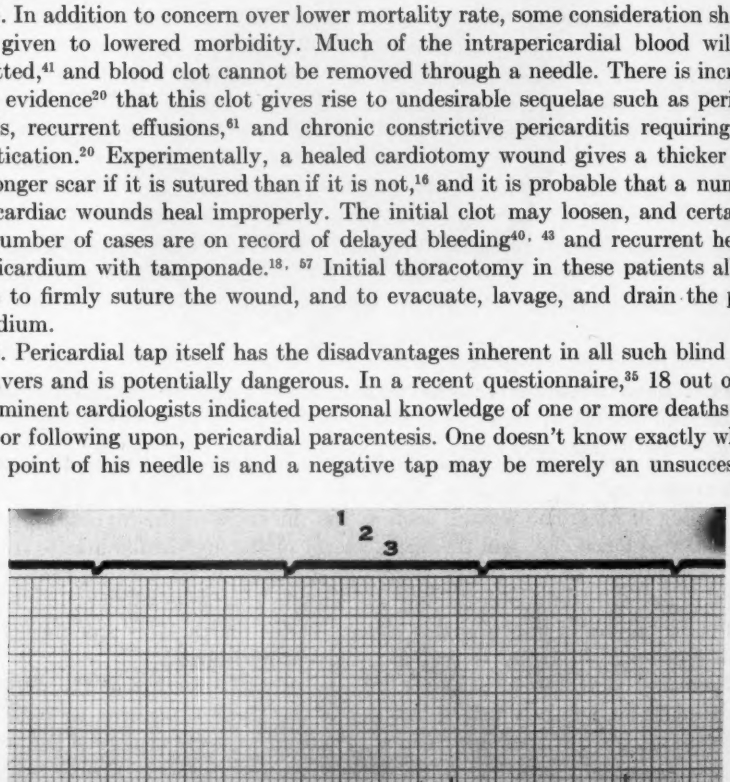


FIG. 4. Electrocardiographic tracing taken while needle encountered ventricular wall during pericardial tap. Note the marked elevation of ST segment.

tap.[45] The needle may puncture or lacerate the myocardium, or worse, may lacerate a coronary artery, with resultant bleeding, infarction, or aneurysm formation.[23] The needle will not remove clotted blood[38, 45] or blood gravitated to inaccessible parts of the pericardial sac.[41, 45] In patients successfully managed by tapping, there is no information as to the site of the wound, or whether it penetrated a chamber. Where possible, we now perform our pericardial taps utilizing the added safety feature of a continuously recording electrocardiogram with the precordial lead attached to the needle. A striking elevation of the S T segment occurs when the needle touches the myocardium[7] (fig. 4).

7. With good preoperative preparation, general anesthesia with an adequate airway, blood volume replacement, and expeditious surgery by an individual versed in cardiac resuscitation, exploratory thoracotomy should entail a minimum of risk even in these critically ill patients. Thoracotomy provides the only method of salvaging those patients who survive to reach the hospital but quickly die of exsanguination. Even under close observation it is a little risky to permit a patient to suffer repeated episodes of acute tamponade, and his over-all physiologic status may be better served by subjecting him to prompt thoracotomy and definitive management of his one or more lesions. There is still some truth in Strieder's[59] statement, "not operating upon a patient with a stab wound of the heart is a very hazardous course to follow."

SUMMARY

Fourteen additional cases are presented of cardiac stab wound in which the patient was treated by thoracotomy, with 2 deaths. Some of the factors favoring an operative approach are discussed.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Anderson, H. M., and Starbuck, R. W.: Conservative therapy of cardiac tamponade resulting from wounds of heart, J. Florida M. A. *33:* 86 (Aug.) 1946.
2. Aristotle (384–322 B. C.): De partibus animalium, Lib. III, cap. 4, Opera edidit Academia Regia Borussica *3:* 328.
3. Beck, C. S.: Wounds of heart; technic of suture, Arch. Surg. *13:* 205 (Aug.) 1926.
4. Bigger, I. A.: Wounds of heart and pericardium, South. M. J. *25:* 785 (Aug.) 1932.
5. ——: Heart Wounds, J. Thoracic Surg. *8:* 239 (Feb.) 1939.
6. ——:Diagnosis and treatment of heart wounds, with summary of 34 cases, M. Ann. Dist. Columbia *9:* 390 (Nov.) 1940.
7. Bishop, L. H., Jr., Estes, E. H., Jr., and McIntosh, H. D.: Electrocardiogram as safeguard in pericardiocentesis, J.A.M.A. *162:* 264 (Sept. 22) 1956.
8. Blalock, A.: Discussion of reference.[5]
9. —— and Ravitch, M. M.: Consideration of nonoperative treatment of cardiac tamponade resulting from wounds of heart, Surgery *14:* 157 (Aug.) 1943.
10. Blau, M. H.: Wounds of heart, Am. J. M. Sc. *210:* 252 (Aug.) 1945.
11. Block: Verhandlungen der Deutschen Gesellschaft fur Chirurgie, Elften Congress, Berlin, Part I, p. 108, 1882.
12. Bode, F.: Versuche uber herzverletzungen, Beitr. Z. Klin. Chir. *19:* 167, 1897.
13. Brockman, H. L., Cooley, D. A., and De Bakey, M. E.: Factors influencing survival in experimental heart wounds, S. Forum *4:* 108, 1953.
14. Cappelen, A.: Vulnus cordis, sutur af hjertet, Norsk. Mag. f. Laegevidensk, *11:* 285, 1896.
15. Cohnheim, J.: Lectures on general pathology, London, New Sydenham Society *1:* 21, 1889.

16. Colin, G. T., Jr., and Ziffren, S. E.: Healing of extensive cardiac wounds, J. Thoracic Surg. 24: 346 (Oct.) 1952.
17. Cooper, F. W., Jr., Stead, E. A., Jr., and Warren, J. V.: Beneficial effect of intravenous infusions in acute pericardial tamponade, Ann. Surg. *120:* 822 (Dec.) 1944.
18. Dejou, L.: Compressive hemopericardium in second month of undiagnosed heart wound, Mem. Acad. de chir. *72:* 352 (June 19) 1946.
19. Del Vecchio, S.: Sutura del cuore, Riforma Med. *11:* 38, 1895.
20. Ehrenhaft, J. L., and Taber, R. E.: Hemopericardium and constrictive pericarditis, J. Thoracic Surg. *24:* 355 (Oct.) 1952.
21. Elkin, D. C.: Diagnosis and treatment of cardiac trauma, Ann. Surg. *114:* 169 (Aug.) 1941.
22. ——: Wounds of heart, Ann. Surg. *120:* 817 (Dec.) 1944.
23. ——: Discussion of references.[5]
24. —— and Campbell, R. E.: Cardiac tamponade; treatment by aspiration, Ann. Surg. *133:* 623 (May) 1951.
25. Elsberg, C. A.: Experimental investigation of treatment of wounds of heart by means of suture of heart muscle, J. Exper. Med. *4:* 479 (Sept.–Nov.) 1899.
26. Farina: Discussion, Zentralbl. f. chir. *23:* 1224, 1896.
27. Farringer, J. L., Jr., and Carr, D.: Cardiac tamponade, Ann. Surg. *141:* 437 (April) 1955.
28. Fisher, G.: Die wunden des herzens und des herzbeutels, Arch. f. Klin. chir. *9:* 571, 1868.
29. Galen (130–200?): Medicorum Graecorum Opera, V. *8:* Lipsiae prostat in officina Libraria car. cuoblochii 1824, De locis affectis, edited by D. C. Kuhn, T. VIII, Lib. V. cap. 2, p. 304.
30. Griswold, R. A., and Maguire, C. H.: Penetrating wounds of heart and pericardium, Surg., Gynec. & Obst. *74:* 406 (Feb.) 1942.
31. Hill, L. L.: Report of case of successful suturing of heart, and table of 37 other cases of suturing by different operators, with various terminations and conclusions drawn, Med. Record, p. 846 (Nov. 29) 1902.
32. Hippocrates: The Genuine Works of Hippocrates, Section 6, Aphorism 18, translated by Francis Adams, New York, William Wood & Co., *2:* 252, 1886.
33. Jobert, J. A.: Reflexions sur les plaies penetrantes du coeur, Arch. Gen. de Med. *6:* 1, 1839.
34. Jordan, P., Jr., and Wilson, G. E., Jr.: Surgical treatment of vascular trauma, S. Clin. North America *33:* 1151 (Aug.) 1953.
35. Kotte, J. H., and McGuire, J.: Pericardial paracentesis, Mod. Concepts Cardiovas. Dis. *20:* 102 (July) 1951.
36. Kuno, Y.: Mechanical effect of fluid in pericardium on function of heart, J. Physiol. *51:* 221 (Sept.) 1917.
37. Linder, H., and Hodo, H.: Stab wounds of heart and pericardium, South. M. J. *37:* 261 (May) 1944.
38. Maguire, C. H.: Discussion of reference.[24]
39. —— and Griswold, R. A.: Further observations on penetrating wounds of heart and pericardium, Am. J. Surg. *74:* 721 (Nov.) 1947.
40. Mason, L. B., Warshauer, S. E., and Williams, R. W.: Stab wound of heart with delayed hemopericardium, J. Thoracic Surg. *29:* 524 (May) 1955.
41. Maynard, A. de L., Cordice, J. W. V., Jr., and Naclerio, E. A.: Penetrating wounds of heart, Surg., Gynec. & Obst. *94:* 605 (May) 1952.
42. Menendez, C. V.: Stab wounds of heart and pericardium producing acute cardiac tamponade, Am. Surgeon *18:* 66 (Jan.) 1952.
43. Mocquot, P., and Constantini, H.: Plaides du coeur a symptomes retardes, Rev. de chir. *58:* 257, 1920.
44. Morgagni, J. B.: De sedibus et causis morborum, Lipsiae, Leopoldi Vossii, 1827–1829.
45. Naclerio, E. A., Maynard, A. de L., and Cordice, J. W. V., Jr.: Personal experiences with 10 consecutive cases of heart wound treated successfully by pericardiotomy and cardiorrhaphy, J. Thoracic Surg. *25:* 448 (May) 1953.
46. Nelson, H.: Penetrating wounds of heart, Arch. Surg. *47:* 571 (Dec.) 1943.
47. Paget, S.: The Surgery of the Chest, London, John Wright & Co., p. 121, 1896.
48. Peck, C. H.: Operative treatment of heart wounds, Ann. Surg. *50:* 100 (July) 1909.
49. Pool, E. H.: Treatment of heart wounds; report of successful cardiorrhaphy and tabulation of cases, Ann. Surg. *55:* 485 (April) 1912.
50. Ravitch, N. M., and Blalock, A.: Aspiration of blood from pericardium in treatment of acute cardiac tamponade after injury, Arch. Surg. *58:* 463 (April) 1949.
51. Rehn, L.: Uber penetrirende herzwunden und herznaht, Arch. f. Klin. chir. *55:* 315, 1897.
52. Rose, E.: Herztamponade (ein beitrag zur herzchirurgie), Deutsche ztschr. f. chir. *20:* 329, 1884.
53. Salomoni: Beitrag zur chirurgie des herzens, Abst. in Zentralbl. f. chir. *23:* 1224, 1896.
54. Sawyer, C. G., Johnston, F. R., and Lambeth, W. A.: Factors in acute cardiac tamponade; altered concept, S. Forum (1951) p. 282, 1952.

55. Schoenfeld, H. H.: Heart injuries, with suture, Ann. Surg. *87:* 823 (June) 1928.
56. Senac, J. B.: Traite de la structure du coeur, de son action et ce ses maladies, Paris, Briasson *2:* 366, 1749.
57. Skinner, H. L., and Farr, K. F.: Decortication of heart, J.A.M.A. *152:* 234 (May 16) 1953.
58. Smith, W. R.: Cardiorrhaphy in acute injuries, Ann. Surg. *78:* 696 (Dec.) 1923.
59. Strieder, J. W.: Stab wound of heart, J. Thoracic Surg. *8:* 576 (June) 1939.
60. Tuffier, T.: La chirurgie du coeur, Cinquieme Congres de la Societe Internationale de Chirurgie, Paris (July 19–23) 1920. Extrait, Brussels, Hayez, 1920.
61. Warren, J. V., Brannon, E. S., Stead, E. A., Jr., and Merrill, A. J.: Pericardial tamponade from stab wound of heart and pericardial effusion of empyema, Am. Heart J. *31:* 418 (April) 1946.
62. Wolff-Wiesinger, L.: Traumatisch entstandenes haemopericard unter dem bilde einer pericarditischen Cirrhose, Ann. paediat. *164:* 209, 1945.

EXPERIENCES WITH AORTIC DISSECTING ANEURYSMS

CHAMP LYONS, M.D., W. STERLING EDWARDS, M.D.

Birmingham

Until quite recently, the diagnosis of dissecting aneurysm of the aorta was an academic exercise of little practical help to the patient. Treatment was symptomatic and expectant, and fatal rupture occurred in 75 to 90 per cent. The small number of survivors from this catastrophe have usually been found to have a dissection of the aneurysm back into the aortic lumen, thus producing a double-barreled channel. The efforts of Gurin, Bulmer and Derby[6] in 1935, Shaw[13] in 1954, and DeBakey, Cooley, and Creech[3] to reproduce surgically Nature's method of re-entry have been encouraging. DeBakey and his associates[3] described a technic of dividing the thoracic aorta between clamps, obliterating the false passage below by approximating the inner and outer layers, excising a window in the intimal and medial layer above and reanastomosing the two ends. Thus a re-entry window from the outer false passage is created into the normal lumen and the distal passage obliterated. It is thought that this procedure will allow a decrease in pressure in the false passage, thus reducing the liklihood of external rupture.

The introduction of a satisfactory method of palliation or cure of dissecting aneurysm makes diagnosis no longer academic but extremely important. Even in the most rapidly progressive form of the disease, hours or days elapse between onset of symptoms and fatal external aortic rupture, allowing sufficient time for emergency diagnosis and treatment.

Three cases are here reported in detail to add additional experience to the small number previously reported in the literature by others and to bring out unusual features which each case presented.

CASE REPORTS

Case 1. A 39 year old aircraft mechanic had been told in 1949 that he had hypertension when he volunteered as a blood donor. One month before admission, during physical exertion he experienced a sudden burning chest pain at the lower part of his sternum, going up into his neck. The pain did not radiate into the arms, back, abdomen or legs at that time. He fell backwards and was unconscious for a few seconds; his wife stated that he had a convulsion. The chest pain lasted for 1 hour and was only relieved by an injection after he was hospitalized. His blood pressure was said to be so high that it could not be measured at that time. That night in addition to chest pain he noted the development of slight tingling and numbness of the right hand, followed by severe pain in the left leg for several hours. One week later, after chest pain had disappeared and ambulation resumed, he noted the onset of marked fatigue in his buttocks and legs especially the calves after walking 50 yards or more, relieved by rest. He had no loss of sexual potency. Two regitine tests were said to be positive but adrenal exploration was deferred and the patient referred to the Veterans Administration Hospital, Birmingham, when a difference in the blood pressure of his arms and legs was discovered. On examination blood pressure was 140/110 in right arm, 210/110

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

in left arm, 135/60 in right leg, 0 in left leg. He appeared well at the time of admission and was having no pain. Funduscopic examination showed tortuous arterioles but no hemorrhages or exudates, disks were normal. The heart was not enlarged, there was a faint systolic apical murmur. The left carotid pulse was faint, the right strong. The right radial pulse was weaker than the left. No pulse could be felt in the left femoral artery and the right femoral pulse was weak.

Laboratory examination: urine specific gravity 1013, hemoglobin 13.5 grams, white blood cells 7,800 per cu. mm. with normal differential, corrected sedimentation rate was 34 mm. per hour. Intravenous pyelograms showed good function bilaterally. Phenolsulfonephthalein test showed 25 per cent excretion and 60 per cent in 2 hours. Electrocardiogram was normal, all blood chemistries were normal except for slight elevation of serum alkaline phosphatase and serum bilirubin. Serology was negative. On Dec. 21, 1955, a retrograde

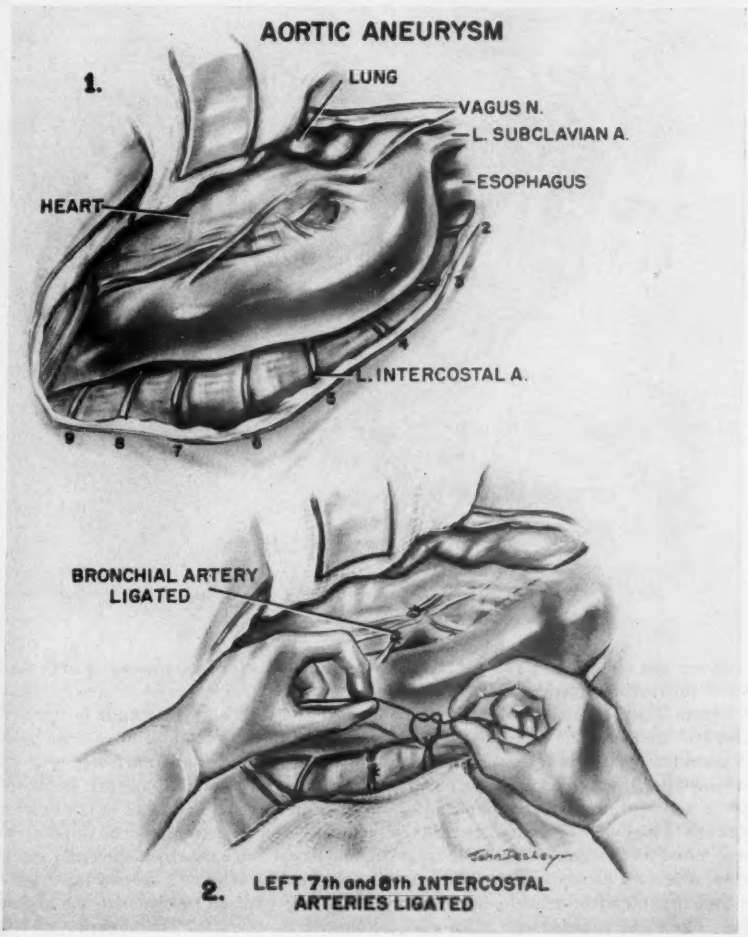


FIG. 1. Technic used in case 1 for establishing a re-entry channel in the thoracic aorta

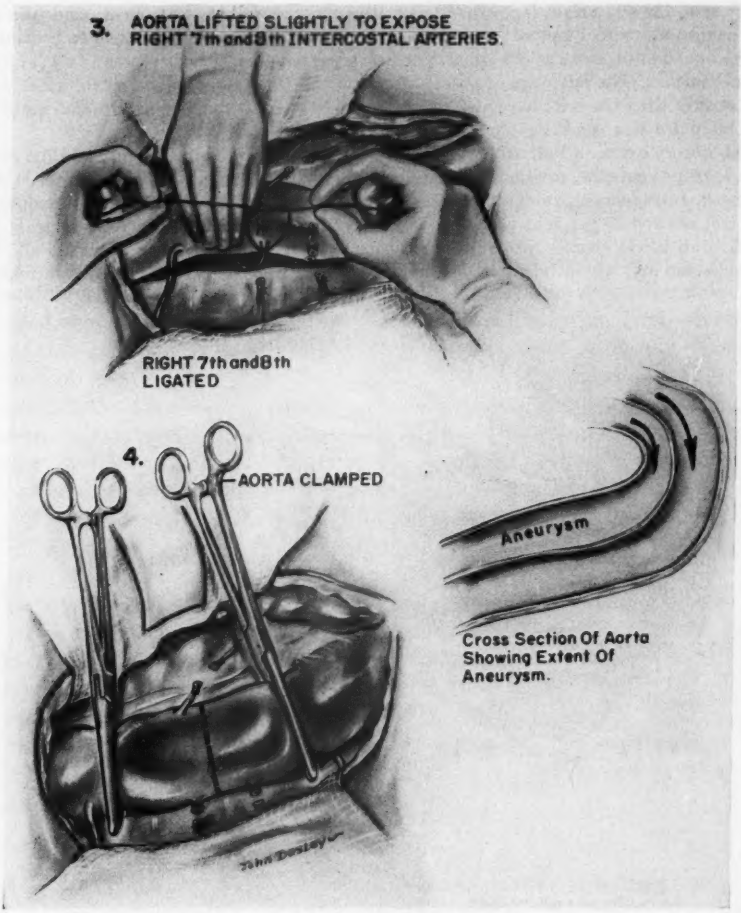

3. AORTA LIFTED SLIGHTLY TO EXPOSE RIGHT 7th and 8th INTERCOSTAL ARTERIES.
RIGHT 7th and 8th LIGATED
4.
AORTA CLAMPED
Aneurysm
Cross Section Of Aorta Showing Extent Of Aneurysm.

FIG. 1. Cont'd.

aortogram was obtained by introduction of a cardiac catheter into the aortic arch through the left subclavian artery. The first injection was made with 30 per cent urokon while the right carotid artery was occluded and the films were taken at $^{5}/_{10}$ seconds intervals. The aortic arch and thoracic aorta were faintly visualized in the left anterior oblique position. The proximal portion of the arch appeared of normal size but rapidly narrowed as it turned downward into the thorax and was never wider than about 1 cm. in diameter. It tapered to as little as 7 mm. toward the diaphragm. Adjacent to this main channel in the descending aorta was a less well visualized channel which composed the remainder of the descending aortic mass. At the level of the diaphragm the main aortic channel widened and reached a normal diameter at the origin of the superior mesenteric artery. A second injection of 70 per cent urokon with pressure cuffs on each thigh was utilized to visualize the abdominal aorta. The right renal artery filled early, followed by the left. The superior mesenteric artery filled but relatively poor visualization was considered to indicate diminished blood

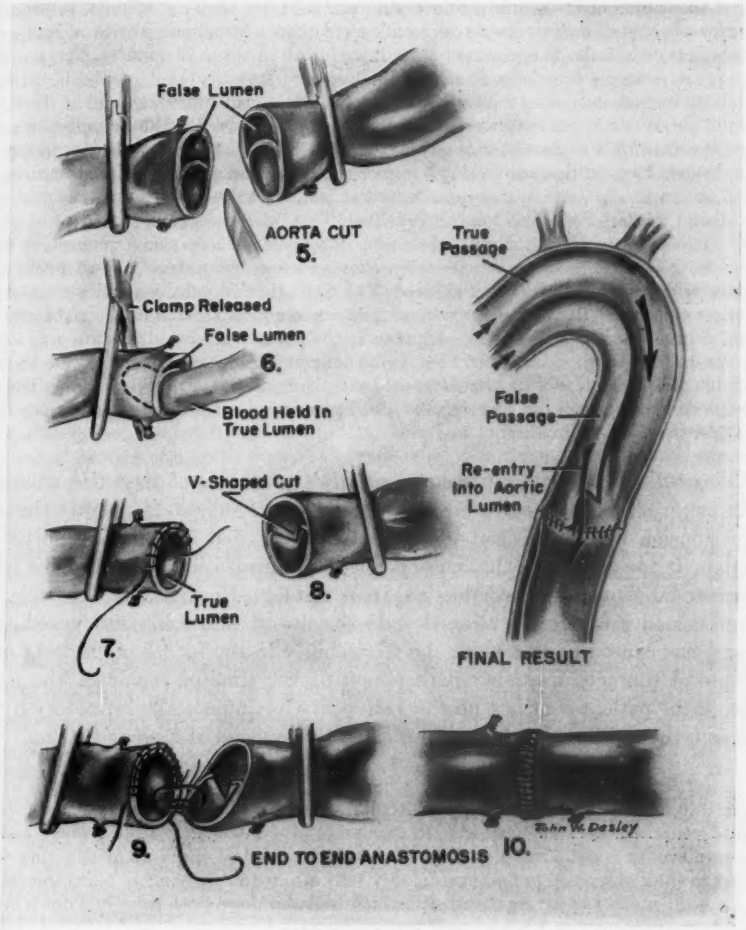


FIG. 1. Concluded

flow. The aorta again narrowed toward the bifurcation and most of the contrast material entered the right common iliac although a small amount could be visualized in the left. It was believed that these films were conclusive of a diagnosis of dissecting aneurysm of the aorta. On December 27, the descending aorta was exposed through the bed of the sixth rib on the left. The arch of the aorta appeared to be about 3 times its normal diameter and presented forceful pulsations posteriorly with a very weak pulsation anteriorly. Two sets of intercostals were divided in the middescending aorta and occluding clamps were applied about 2 inches apart (fig. 1). The aorta was completely transected between these two clamps and a false cannel was found dissecting about ⅓ of the circumference of the aorta anteriorly. This false channel was allowed to collapse inferiorly by momentary release of the distal clamp following which the dissected intima was resutured to the media and adventitia by a completely circumferential continuous suture of no. 0000 arterial silk. About 1 square

cm. of the intima of the segment above was resected from the false channel to prepare a re-entry channel. The two aortic segments were then approximated with a continuous running suture and the clamps were released. Occlusion time was 23 minutes. This patient's postoperative course was uneventful. Good pulses were palpable at all arteries in the lower extremity immediately after surgery but the left carotid remained weak and he developed signs of persistent hypertension several days after operation with systolic pressures measuring over 200. He was started on reserpine. On Jan. 2, 1956, a further medical work-up was undertaken. Two regitine tests using 5 mg. of the drug intravenously failed to reduce the blood pressure. The section of aorta removed at operation showed some fragmentation of the elastic tissue adjacent to the false channel. This false passage appeared to be partly epithelialized with the epithelium lying on a thin layer of loose young connective tissue cells. No mucinous or other degenerative changes were demonstrable. Two weeks after operation the patient appeared stabilized. The right carotid pulse was still considerably stronger than the left. No pulses or blood pressure were obtainable in the right arm but arterial circulation appeared to be adequate. In the left arm the blood pressure was 165/90. He was discharged on reserpine of 2 mg. twice daily and moderate salt restriction. A letter from his physician in another city, 3 months after operation revealed that the patient was doing well symptomatically but that his blood pressure was continuing high in the range of 200/90 despite continuation of reserpine.

Comment: This patient represented a fairly typical case of dissecting aneurysm with confirmation of the diagnosis by aortography. As is so frequently the case, hypertension was an associated problem and preceded the onset of aortic dissection. It seems certain that the operation performed has improved the circulation to his kidneys, gastrointestinal tract and legs. Nevertheless, the continued severe blood pressure elevation despite the use of Rauwolfia and vasodilating drugs, has caused us to ponder the advisability in the future of carrying out a unilateral thoracolumbar sympathectomy at the time of repair of the aorta. Should the patient continue postoperatively to have medically refractory hypertension, the right sympathetic chain could be removed at a second stage.

Case 2. This patient was a 55 year old white retired railroad engineer. He had not worked since 1949 because of asthmatic bronchitis but had been relatively asymptomatic for the past 2 years. About 7 months prior to admission he fainted after feeling dizzy and was unconscious for about 5 minutes. He had a complete medical check-up at this time which failed to show any positive findings. In July 1955, while driving home in his automobile he had a sudden onset of severe chest pain which radiated down both arms and down his left leg. He drove some 6 or 7 blocks to a doctor who gave him a "*shot*" and referred him to a local hospital in an ambulance. The pain was very severe from the time of onset but continued to increase in severity and he felt cold and extremely short of breath before he received an injection. He was placed in an oxygen tent in the hospital; a diagnosis of coronary thrombosis was made and he was placed on dicumarol. He remained in that oxygen tent for 6 days and pain recurred when he was taken out of the tent, so he was placed there for 2 more days. This hospital admission lasted 3 weeks and he remained in bed for 5 weeks after returning home. During this time he continued to have shooting pain in his left chest lasting from 1 to 20 minutes. These were associated with throbbing pain in his temples and a feeling of compression in his head. Any exertion caused the pain in his chest to radiate into his arms and down his left leg. Two weeks before his admission he was sent to his family doctor for a check-up and he was found to have a blood pressure of 110/70 in his left arm and 170/90 in his right arm. About this time he first noted a pain in the back in the left subcapular area but most of his back pain was lumbar and bilateral. This became

more severe along with increase in the chest pain the night of admission and this caused him to apply for hospitalization. Nitroglycerin failed to relieve this pain. On admission he denied any history of syphilis, hypertension, or diabetes. On examination the blood pressure in the right arm was 170/90, left arm 110/80. The right carotid pulse was normal, the left carotid pulse weak. There was a systolic bruit which could be heard in the region of the umbilicus. The left radial pulse was decreased as compared with the right. The left femoral pulse was absent and the right was very much diminished. Popliteal, posterior tibial and dorsalis pedis pulses were absent bilaterally. Laboratory data: urinalysis negative, white blood cell count 7,900 with a normal differential, hemoglobin 12 grams, blood urea nitrogen 19.5, electrocardiogram normal, P.S.P. test revealed 3 per cent excretion in 15 minutes, 15 per cent excretion in 30 minutes. Chest roentgenograms shortly after admission in several views revealed no abnormality. There was no increase in size in the aortic shadow and no demonstrable calcium in the wall of the aorta. A film of the abdomen revealed a small amount of calcium in the iliac vessels adjacent to the sacrum but no calcium was seen in the abdominal aorta. Because of a history of allergy to iodine no aortography was advised on this man and it was thought by a combined surgical-medical cardiovascular review that he had variable physical signs which could best be explained on the basis of dissecting aneurysm of the aorta. Accordingly on Feb. 15, 1956, the descending aorta was exposed through a left chest incision through the bed of the sixth rib, three sets of intercostals were divided in the mid-descending aorta and the aorta was cross clamped with a segment about 2 inches in length between clamps. Transection was carried out and no dissecting aneurysm was found. Several internal plaques along the lumen were identified, the two ends of the aorta were sutured together and the clamps were released with an occlusion time of 16 minutes. This patient's postoperative course was uneventful. It was thought in retrospect that he had had obstruction of the left subclavian artery and possibly of the left carotid by atheromatous plaques or arteriosclerotic narrowing with obliterative arteriosclerotic obstruction of the abdominal aorta or common iliac vessels.

Comment: This case is reported to stress the value of angiography in preventing the error that was made here. The only finding in this man's work-up not entirely consistent with dissecting aortic aneurysm was the absence of widening of the thoracic aortic shadow on roentgenograms of his chest.

Case 3. This 37 year old Negro laborer was asymptomatic until 1 month prior to hospital admission. He had been turned down for Army service after spending 1 day at an Army base, but he was not told the reason why. This was in 1942. One month before admission while sitting at rest he suddenly was seized by a severe pain in the left side of his chest. This pain rapidly extended to the left shoulder, left side of the back and down the left arm to the elbow; down the left flank to the left hip. He broke out in a sweat but did not become nauseated or faint. The pain was extremely severe and persisted without much let up for at least 6 hours before he went to sleep that night. The following day the pain was better and it gradually became less severe in the next 2 weeks, with an occasional exacerbation unrelated to exercise. There was no history of syphilis or other venereal disease. He had had repeated blood tests in the past; the last one being July 1954 and these had always been reported negative. He had never received any heavy metal or penicillin therapy. There was no history of numbness, pain tingling, or claudication of the extremities, nor history of hoarseness or hemoptysis. On physical examination a grade three systolic murmur could be heard over the entire precordium, but loudest in the pulmonic area. It was heard with equal intensity along the medial border of the left scapula in the back. The aortic second sound was not remarkable. Blood pressure in the right arm was 160/90 and the left arm 172/100. The femoral pulses were barely palpable bilaterally and there was no pulse below this. Blood pressure in the legs was not obtainable. The E.K.G. findings were compatible with a complete right bundle branch block. Roentgenograms of the chest and

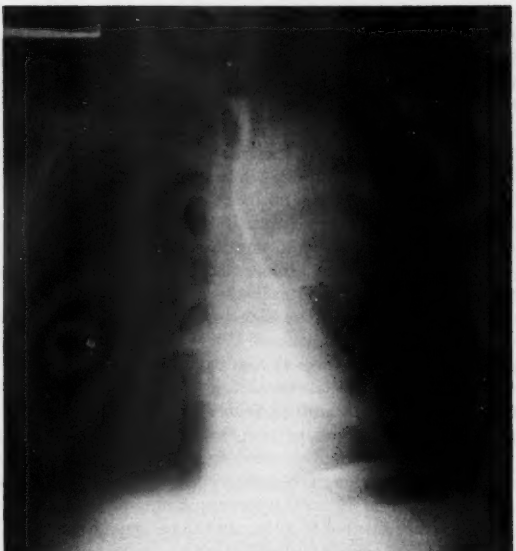


FIG. 2. Chest roentgenogram in case 3 showing wide descending aorta distal to coarctation.

FIG. 3. Specimen opened in case 3 showing area of coarctation and dissecting aneurysm distal to coarctation.

fluoroscopy revealed a large descending aorta which seemed to extend up into the arch and down to the dome of the diaphragm (fig. 2). There was very little pulsation of this mass, a preoperative diagnosis of dissecting aneurysm distal to a coarctation was made, and the patient was operated upon April 4, 1955. At operation a constriction was found 2 cm. distal to the left subclavian artery, with a huge poststenotic aneurysm extending almost to the diaphragm. The coarctation and aneurysm were freed up and the descending aorta resected from 1 cm. below the subclavian to just above the diaphragm. The defect was replaced with a lyophilized homograft 7 inches long sutured end to end, with an occlusion time of 60 minutes. This patient's postoperative recovery was uneventful, his femoral and pedal pulses were immediately restored and his arm blood pressures returned to normal. At the end of 18 months he is doing well and has returned to work as a truck driver.

The opened specimen (fig. 3) of resected aorta showed a transverse split in the aortic intima approximately 3 cm. below the coarctation with a dissected channel extending for approximately 10 centimeters. The orifice of the coarctation measured 3 millimeters in diameter. The entire area of dissection had been removed.

Comment: Approximately one fourth of the reported deaths from untreated aortic coarctation result from rupture.[5] Reifenstein and associates[12] in reviewing 104 autopsied cases of coarctation found 24 cases of death from rupture. Of these disruption occurred proximal to the constriction in the ascending aorta in 19 cases, and distal to the coarctation in 5 cases. Rupture rarely occurs immediately proximal to a coarctation. The average age at death from this cause was found to be 27 years.

A thorough review of the literature has revealed a total of 9 cases[12, 14, 4, 11, 1] of rupture of the thoracic aorta distal to the coarcted area including the 5 mentioned above. Hemorrhage occurred into the left plural cavity[14] bronchus[4, 11] or esophagus.[1, 11] Rupture was almost always rapidly fatal, but there are 2 reported cases which suggest the possibility of survival. Hecker[7] reported the case of a 62 year old man with sudden severe back pain, so severe that he shot himself 12 days later. Autopsy revealed a coarctation 2 mm. in diameter with an aneurysm below. Six inches below the constriction there was a longitudinal slit in the posterior wall with dissection up to the coarctation, and downward to the bifurcation of the aorta. The second case[12] was that of a 45 year old man dying from congestive failure 19 months after dissection of an aneurysm distal to a moderate coarctation. Autopsy revealed that the aneurysmal lumen was endothelialized and contained a canalized thrombus. There have been no previous cases reported of surgical cure of such a postcoarctation aneurysm after rupture or dissection has occurred.

SUMMARY

The cases of 3 additional patients operated upon for dissecting aneurysm of the thoracic aorta are reported to add to the very small number previously reported.

One patient with typical history and findings proved not to have a dissection. This points out the need for improved diagnostic methods, especially aortography, in these cases.

A case of dissecting aneurysm distal to a coarctation of the aorta is reported.

This is thought to be the first successful cure of this rare type of dissecting aneurysm.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Bargi, L.: Contributo allo studio delle stenosi aortiche dell 'arco, Clin. med. ital *65:* 152 (Feb.) 1934.
2. Barsantini, J. C., and Bazzano, J. J.: Estenosis del istmo de le aorta, Arch. urug. de med., cir. y especialid. *13:* 448 (Oct.) 1938.
3. De Bakey, M. E., Cooley, D., and Creech, O., Jr.: Surgical considerations of dissecting aneurysms of aorta, Ann. Surg. *142:* 586 (Oct.) 1955.
4. Goodson, W. H., Jr.: Coarctation of aorta; report of 2 unusual cases, New England J. Med. *216:* 339 (Feb. 25) 1937.
5. Gross, R. E.: Coarctation of aorta, surgical treatment of 100 cases, Circulation *1:* 41 (Jan.) 1950.
6. Gurin, D., Bulmer, J. W., and Derby, R.: Dissecting aneurysm of aorta; diagnosis and operative relief of acute arterial obstruction due to this cause, New York J. Med. *35:* 1200 (Dec. 1) 1935.
7. Hecker, J. T.: Coarctation of aorta, report of case with rupture distal to constriction, J. Iowa M. Soc. *29:* 240 (June) 1939.
8. Kriegk, M.: Drci neve falle van stenose der aorta in der gegend der insertion des ductus botalli, Vrtljschr. f.d. prakt, Heilk., Prague *47:* 137, 1878.
9. Lendet, E.: Recherches anatomiques et cliniques sur le retrecissement de l'aorta an novean de canal arteriel, Gaz. Med. de Paris *13:* 44, 1858.
10. Monckeberg, G.: Enige komplikationen. bei ange norener stenose des isthmus der aorta, Deutsch. path. Gesellsch. *11:* 267, 1907.
11. Moragues, V., Moore, L. T., and Rossen, J. A.: Coarctation of aorta with rupture of wall below point of constriction, Am. Heart J. *24:* 828 (Dec.) 1942.
12. Reifenstein, G. H., Levine, S. A., and Gross, R. E.: Coarctation of aorta; review of 104 autopsied cases of adult type, Am. Heart J. *33:* 146 (Feb.) 1947.
13. Shaw, R. S.: Acute dissecting aneurysm treated by fenestration of internal aneurysmal wall, New England J. Med. *253:* 331 (Aug.) 1955.
14. Zaslow, J., and Krasnoff, S. O.: Coarctation of thoracic aorta with aneurysm distal to obstruction, Am. Heart J. *26:* 832 (Dec.) 1943.

CAROTID-CAVERNOUS ARTERIOVENOUS ANEURYSM*

J. Garber Galbraith, M.D.

Birmingham

INTRODUCTION

Unilateral pulsating exophthalmos due to carotid-cavernous aneurysm was first described by Benjamin Travers in 1809.[3] The clinical aspects of this lesion have been widely recognized and well documented, requiring no reiteration here.[4] The purpose of this report is to present a rationale of treatment based on consideration of the anatomic and physiologic principles involved with these lesions, and the associated alterations in cerebral hemodynamics. Selected case reports are presented, indicating that a regimen of surgical management based on these principles affords effective control of this lesion with minimal risk.

ANATOMIC CONSIDERATIONS

Due to the anatomic peculiarities of this region, the ideal treatment of arteriovenous aneurysm, namely quadruple ligation and excision, is manifestly inapplicable. Except in unusual cases it has further been demonstrated that ligation of the venous tributaries of these lesions is neither necessary or desirable. Occlusion of all or most of the arterial supply is generally sufficient to induce obliteration of the fistula by thrombosis. The arterial supply is from the internal carotid artery, and collateral flow is from the ophthalmic artery and the intracranial portion of the internal carotid artery by reflux flow. Arteriographic studies have demonstrated that usually most, if not all, of the blood flowing into the fistula via the proximal internal carotid artery is shunted through the fistula. One may then assume that adequate collateral cerebral circulation is available in the untreated patients since the blood flow through the internal carotid on the involved side is shunted into the fistula and little or none of this blood reaches the brain. Ligation of the internal carotid artery in the neck, however, alters the intracranial hemodynamics by lowering the pressure in the fistula, thus tending to induce reflex flow into the fistula from the intracranial carotid trunk. This may produce cerebral ischemia with possible disastrous results. Similar alteration of direction of blood flow may occur in the ophthalmic artery. A preliminary Matas test is of great value in detection of possible cerebral and retinal ischemia due to reflux flow into the fistula induced by cervical carotid occlusion. In the presence of an arteriovenous fistula, ligation of the carotid is tolerated less well than with sacular aneurysms, as noted by several observers.[5] Inability of the patient to tolerate temporary carotid occlusion may thus indicate disruption of previously adequate collateral circulation due to induced reflux intracranial carotid and ophthalmic artery flow. The implication

* Presented in part at Southern Neurosurgical Society, Baltimore, Jan. 30, 1954.
From the Division of Neurosurgery, Department of Surgery, Med. College of Ala., Birmingham, Alabama.

of this finding is evident; the intracranial portion of the internal carotid artery distal to the aneurysm, and the ophthalmic artery at its origin, should be occluded before ligating the cervical portion of the internal carotid artery, when the Matas test indicates inability to tolerate cervical carotid occlusion.

That the internal carotid artery is the vessel to be occluded in the neck is obvious. Common carotid ligation does not effectively stop blood flow through the internal carotid artery, and this flow may be in either direction, varying in individual patients. The external carotid artery should be spared from ligation, as emphasized by Botterell,[1] since its anastomotic branches furnish blood to the optic nerve and retina via the ophthalmic artery, after ophthalmic artery ligation at its point of origin. Blindness which may otherwise occur is thus rendered less likely. That the ophthalmic artery may contribute significantly to the aneurysm and thus must be ligated, is indicated by repeated observation of the efficacy of external carotid ligation in relieving symptoms of the fistula where cervical internal carotid ligation alone was inadequate[7] and where no intracranial procedure had been done. The greater risk of blindness, however, renders this method of management undesirable.

CASE REPORTS

Case 1. B. R., Negro man aged 58, was first admitted to the Lloyd Noland Hospital on July 13, 1949 and dismissed on July 25. He complained of left sided headache, pain in left eye and roaring in left ear since January 1949. He sustained a crushing injury of left side of his face in October 1928.

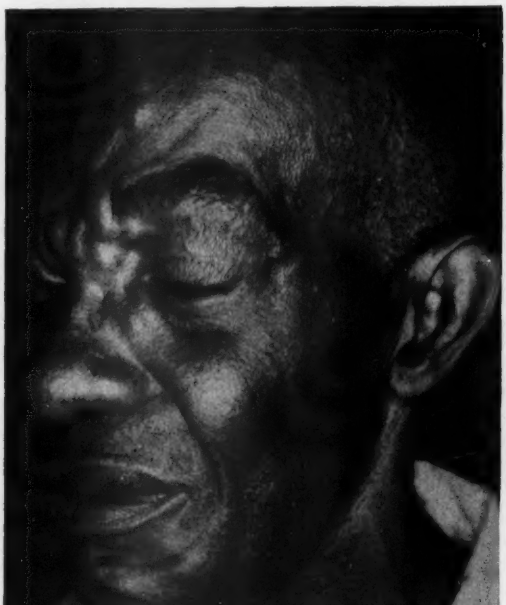


FIG. 1. B. R. Engorged facial veins with arterial pulsation. Proptosis with pulsation of eye.

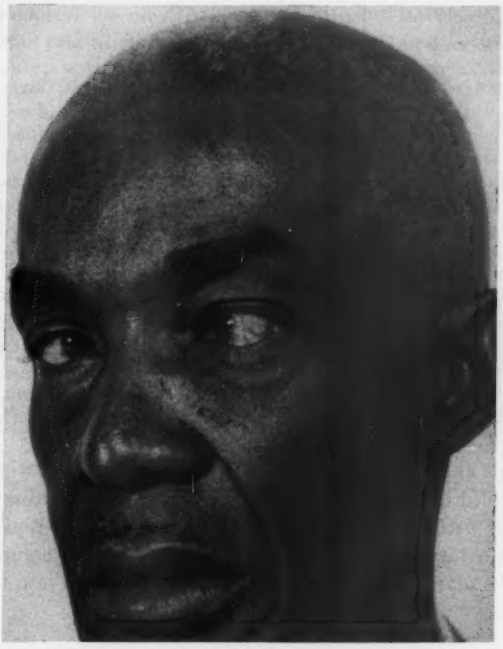


FIG. 2. B. R. Postoperative, after combined craniotomy and cervical ligation. All eye signs have cleared except bilateral. N. VI palsy.

Examination: Proptosis, left eye, pulsating. Very prominent orbital and frontal veins. Vision poor. Bruit and thrill over eye, obliterated by left carotid compression (fig. 1).

Operation: July 19: Left common carotid artery constricted by tantalum band until bruit ceased. Wound closed. Patient became comatose after 10 minutes. Wound opened and tantalum band removed. Patient began to respond after 4–5 minutes.

His second admission was on November 30 and dismissal was on Dec. 9, 1949. His headaches had become severe and his vision worse.

Operation: December 6: Left common carotid artery constricted 80 per cent by tantalum band. Bruit reduced.

His third admission was on July 11, 1951 with dismissal on July 25. His headaches had again become severe. He would not tolerate complete compression of left carotid more than 3 minutes.

Operation: July 13: Left frontal craniotomy, internal carotid artery occluded above cavernous sinus. Ophthalmic artery coagulated. Left internal carotid artery completely ligated in neck. Bruit ceased. Prominent veins disappeared. Headache relieved. Vision improved.

Follow-up March 1956. Well, except residual bilateral N. VI paralysis. Vision good (fig. 2).

Comment: After a 2 year effort to occlude the carotid in the neck, it became obvious that this would never be tolerated. Coma, convulsions and hemiplegia followed this procedure, relieved by restoration of carotid flow. After the intracranial trunks communicating with the fistula were ligated, the internal carotid

artery was completely occluded in the neck with no deleterious effect and with complete subsidence of proptosis, orbital pulsation, bruit and pain.

Case 2. L. S., Negro woman aged 75, was first admitted to St. Vincent's Hospital on Aug. 21, 1951 and was dismissed on August 31. She gave a history of blindness. She woke up 2 weeks ago with headache, roaring noises in right ear and poor vision. Both eyes remain closed, and if opened passively vision was very poor in right eye. She was blind in left eye.

Examination: Alert. Blood pressure was 240/115. Bilateral pulsating exophthalmos with ptosis. Both eyeballs in a fixed position. Perceives objects dimly with right eye. Blind left eye with haziness of cornea. Orbital and frontal veins prominent, with bruit and thrill over entire area, obliterated by compression of right common carotid artery. Would not tolerate compression more than two minutes.

Operation: August 27: 1. Right frontal craniotomy. Internal carotid artery occluded by silver clips above cavernous sinus. Ophthalmic artery cauterized. Bruit sill present. 2. Right internal carotid artery ligation in neck by tantalum band. Bruit ceased.

Course: Ambulatory on August 28. Headache relieved. Exophthalmos and bruit relieved. Vision improved in right eye. Left eye blind (glaucoma).[2] Report from local physician January 1956 indicated that patient was in good health except for hypertension.

Comment: This elderly individual with severe hypertension would not tolerate preliminary carotid compression in the neck. The intracranial trunks communicating with the fistula were therefore occluded initially, and thereafter the internal carotid artery was easily ligated in the neck with complete subsidence of the symptoms attributable to the fistula. There was no untoward neurologic after-effect.

DISCUSSION

These case reports demonstrate the importance in selected cases of initial intracranial ligation of the carotid distal to the fistula in order to protect against cerebral ischemia when the carotid is occluded in the neck. This order of attack has been employed when preliminary Matas test gave evidence of inadequate cerebral collateral flow. Ligation of the ophthalmic artery at its origin likewise tends to prevent embarrassment of retinal blood flow and obliterates the other source of blood to the fistula. Sufficient experience is not yet available to warrant positive opinion, but one might reasonably expect ophthalmic artery ligation to reduce the relatively high incidence of visual impairment reported in cases controlled by cervical carotid ligation alone.[6]

SUMMARY

Management of carotid-cavernous arteriovenous aneurysm consisting of an orderly regimen of measures is outlined, part or all of which may be required for control of the lesion. The simplest and least hazardous measures are instituted first. A small percentage of patients recover spontaneously or are relieved by intermittent digital compression of the carotid artery in the neck. Most patients will require surgical management. If the Matas test is well tolerated, then ligation of the cervical portion of the internal carotid artery is indicated. When this alone is not effectual it should be followed by the intracranial procedure. If preliminary carotid compression or ligation is not tolerated, then the

intracranial ligation of the internal carotid and ophthalmic arteries should be the first order of treatment, following which the internal carotid artery in the neck may be ligated with impunity, completing the surgical control of the aneurysm.

Since a single observer encounters relatively few cases, much wider experience and longer follow-up studies will be required to evaluate the results of this method.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Botterell, E. H : Personal communication.
2. Crawford, H. E.: Carotid-cavernous aneurysm, Arch Ophth. *27:* 539 (March) 1942.
3. Locke, C. E., Jr.: Intracranial arterio-venous aneurysm of pulsating exophthalmos, Ann. Surg. *80:* 1 (July) 1924.
4. Martin, J. D., Jr. and Mabon, R.: Pulsating exophthalmos (arteriovenous aneurysm of cavernous sinus and branches of internal carotid artery): review of all reported cases, J.A.M.A. *121:* 330 (Jan. 30) 1943.
5. Olivecrona, H.: Ligature of carotid artery in intracranial aneurysms, Acta. chir. scandinav. *91:* 353, 1944.
6. Raaf, J., and Swan, K.: Traumatic carotid-cavernous sinus aneurysm, Am. J. Surg. *80:* 816 (Nov. 15) 1950.
7. Wold, K. C.: Carotido-cavernous fistula, Minnesota Med. *24:* 468 (June) 1941.

PERIPHERAL ARTERIAL EMBOLISM

J. M. Donald, M.D., S. J. Campbell, M.D., W. N. Viar, M.D.

Birmingham

Arterial embolism is an age old and intriguing problem to the physician, yet it continues to be a life endangering problem to the patient.

It was over 100 years ago that Virchow[6] elucidated both anatomically and clinically the embolic mechanism. Arterial embolectomy was attempted as early as 1895 but it was not until 1911 that Labbé and later in the same year Key[5] performed the first successful embolectomies.

Arterial embolism remains a life endangering disease because these patients are almost always critically ill from other causes at the time the embolus occurs.

With each passing year great strides are being made in the surgical correction of many vascular abnormalities and certainly a great deal of progress has been made in the care of the patient with arterial embolism. Thus a recent review by Warren, Linton, and Scannell[8] of 200 patients with arterial embolism reveals that the limb survival rate in a group considered between 1927 and 1946 was 66 per cent. A similar group treated between 1946 and 1953 revealed an 86 per cent limb survival rate. In the same two groups the mortality rate was lowered from 35 per cent to 27 per cent.

We have recently treated 2 patients with this interesting malady. Both patients survived but there were several puzzling therapeutic facets presented in their care. It is for this reason this report is being made.

Case 1. G. S., a 70 year old white man was known to have arteriosclerotic heart disease and had developed auricular fibrillation in January 1954. He had been followed as an outpatient receiving Digitalis and Quinidine. At 5 a.m. on Aug. 9, 1954, he developed sudden pain in the right thigh followed shortly by numbness in the right thigh, leg, and foot. The patient was hospitalized at approximately 10 a.m. He was in rather severe distress and was perspiring profusely. The blood pressure was 120/80. The precordial pulse revealed a marked irregularity with a rate of 110 and a radial pulse deficit of 20 beats per minute. The right lower extremity was slightly enlarged, cold, pale, and moist. There was a faint femoral pulse just below Poupart's ligament. Pulsations were absent in the popliteal, posterior tibial, and dorsalis pedis areas. There was slight cyanosis of the toes of the right foot.

After surgical consultation a diagnosis of arterial embolus lodged at the bifurcation of the superficial femoral and profunda femoris arteries was made.

At approximately 12 o'clock noon the patient was operated upon and an embolus with a propagating thrombus was removed from the common femoral artery at its bifurcation. The arteriotomy site was closed intima to intima with no. 00000 arterial silk. Almost immediately after extracting the embolus there were demonstrable pulsations in the distal arteries.

The patient's convalescence was entirely uneventful except for persistent pain in the foot and calf for approximately 48 hours. At no time, however, did the extremity become cold or discolored.

Case 2. D. D., a 64 year old white woman was admitted to the hospital on Jan. 20, 1955,

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

with small bowel intestinal obstruction secondary to tuberculous enteritis. A portion of the ileum was resected. The patient was to be discharged on the fourteenth postoperative day when she developed paroxysmal auricular tachycardia which persisted for 18 hours despite therapy. During this episode, the patient experienced squeezing substernal pain. This persisted for about 1 hour when it was relieved with Nitroglycerine and Pantopon. She was digitalized the next day and allowed to go home the following day.

Three days later, at approximately 10 a.m., the patient developed sudden pain in the calves of both extremities followed very soon by burning in the heels and soles of the feet. Symptoms progressed very rapidly and after about 2 hours there was almost complete paralysis of both lower extremities. She was readmitted to the hospital approximately 5 hours after onset.

Examination revealed the patient to be in severe distress complaining of excruciating pain in both lower extremities. Both femoral pulses were absent. The lower extremities were cold and pale with a cadaveric appearance. A diagnosis of saddle embolus at the aortic bifurcation was made. While preparing the patient for surgery, the pulse returned in the left femoral area. The extremity became warm and of normal color and the pain disappeared. The right side remained status quo.

At surgery, the right common iliac vessel was seen to be distended at its bifurcation. Pulsations were absent below this point. The aorta and the iliac arteries were mobilized above and the right femoral artery was mobilized below. These arteries were secured and the right common iliac was opened through a longitudinal incision. An embolus 2½ cm. in length was removed. Intima to intima closure with no. 00000 arterial silk was carefully done. Examination of the femoral artery revealed a spastic segment several cm. in length although there was a fairly strong pulse.

Following surgery, the right leg and foot remained cool and white. Coolness also returned to the left foot; and approximately 12 to 14 hours following surgery, there was a mottled bluish appearance to the right foot and toes and some bluishness of the left heel. Intravenous priscoline was given without improvement. Heparin was begun. The patient was complaining bitterly of pain. Approximately 20 hours following surgery, a long-acting spinal anesthetic was given. There was no improvement for approximately 3½ hours, but at this time, there was rather dramatic improvement in both lower extremities. The left side became normal and the right lower extremity became warm to the ankle and all the discoloration disappeared except for portions of the toes. Approximately 5½ hours following the spinal anesthetic all previous signs and symptoms began to return. At this time, a polyethylene catheter was placed in the subarachnoid space and a continuous spinal anesthetic of dilute Procaine solution was started in a slow drip. Improvement was noted within about 15 to 20 minutes.

The spinal anesthetic was maintained for a period of 9 days at a rate sufficient to maintain sympathetic paralysis and yet show little disturbance in cutaneous sensibility and no disturbance in muscular modality. On the seventh day the Procaine drip was gradually slowed, but the patient began to complain of pain and it was noted that she had developed another episode of paroxysmal auricular tachycardia. The Procaine solution was therefore allowed to drip at its usual rate again and the tachycardia was ended by several trials of vagal stimulation. The patient was placed on Quinidine, and on the eighth day the spinal drip was slowed without mishap and on the ninth day it was discontinued.

The patient continued to have severe pain in the right leg and foot and the toes became gangrenous. On the twentieth postoperative day a low amputation was attempted but because of edema and very little blood supply in the lateral leg muscles, a supracondylar amputation was necessary. Pathologic examination revealed the anterior tibial artery to be the seat of extensive thrombosis. The patient's further convalescence was uneventful.

DISCUSSION

Auricular fibrillation, myocardial infarction with mural thrombosis and bacterial endocarditis constitute the principal causes of arterial embolism. Rarer

causes include paradoxical embolism through a patent intracardiac septum, dislodgement of an arteriosclerotic plaque and tumor embolism. The first patient herein presented had auricular fibrillation. It is postulated that the second patient had endocardial damage with mural thrombosis as a result of the paroxysm of auricular tachycardia. Digitalis following this probably served to dislodge the thrombus by virtue of increased strength of contractility.

Once embolization has occurred, the distal arterial tree becomes spastic which allows stagnation of blood thus creating an ideal situation for arterial thrombosis. Whether the spasm is reflex, myogenic, or both is an unsettled question.[6] It seems likely that each plays a role since both sympathectomy and local application of Papaverine act to relieve the existing spasm.[4]

It is well known that distal arterial thrombosis due to spasm and reduced blood flow is one of the greatest deterrents to successful embolectomy. Shaw[5] has recently reported excellent results, utilizing a very aggressive approach to this problem. He believes that following embolectomy the artery should be exposed distally and "*backwashed*" from the distal point to the primary arteriotomy in order to be sure that any distal thrombus is completely removed. In this manner, one is certain that good arterial flow is re-established. We believe that this is an excellent point and agree with him that it should receive wider usage. If this method had been used in our second case, a good result might well have been obtained.

The great majority of emboli are to limb arteries, over 75 per cent in one series.[3] Approximately 50 per cent involve the main lower limb arteries. The femoral arteries receive the embolus in about 25 per cent of instances. This area being so readily accessible to surgical exploration makes this point tremendously significant.

Probably the single most important improvement made in the treatment of this disease in the past several years is earlier referral of the patient to the hospital. In Linton's[3] series only 28 per cent of patients in group I were referred within 10 hours; whereas 50 per cent in group II were hospitalized within 10 hours. Both of the patients herein presented were operated upon within 10 hours.

A very important consideration, which arose to some degree in the first patient presented and was of paramount importance in the second patient, was that of persistent arterial spasm. Our first patient continued to have some pain for approximately 48 hours although there was never any doubt about the arterial sufficiency of the extremity since it remained warm and of good color. In the second patient presented, the paleness and coolness of the right extremity did not change following surgery and, indeed, there was some deterioration of the circulation to the left extremity.

Spasm occurs not only as a result of the embolic process, but as has been pointed out by Kinmouth, Simeone, and Perlow[2], trauma may cause severe spasm. Edwards and Lyons[1] have also recently pointed out some of the perplexing problems that may be presented following traumatic injuries. It is impossible to avoid trauma at the time of surgery in dissecting out the vessels. We believe

that many lives and even more limbs are lost because of the difficulty of relieving persistent arterial spasm postoperatively.

Continuous spinal anesthesia was used in our second patient by inserting a plastic catheter into the spinal canal and running a continuous drip of dilute Procaine solution. The anesthesiologists performed this procedure which has been little used for this purpose. It is emphasized that this drip was not started for 24 hours following surgery which was too late to save the patient's extremity but probably saved her life. We believe that had the drip been started at the time of surgery and continued, we would have saved this patient's extremity; but as pointed out above, it has been little used and it was not thought of at the time. As can be seen from our case history, one can easily tell when spasm has been relieved and it is also possible to maintain sympathetic interruption without losing muscular modality or cutaneous sensation. There was no evidence at any time of spinal contamination or other untoward symptoms even after 10 days. We believe very strongly that this procedure should receive serious and early consideration in dealing with this problem.

It is generally agreed that embolectomy is the procedure of choice for aortic, iliac and femoral emboli. Linton[3] emphasized six therapeutic principles: (1) early operation, (2) direct and adequate exposure of the embolic site, (3) occlusion of the artery distal to the embolus before manipulation at/or proximal to the embolic site, (4) avoidance of intimal damage, (5) complete control of the arterial inflow both proximal and distal to the arteriotomy, (6) meticulous intima to intima closure. We would like to add a seventh and eighth therapeutic principles: (7) Where there is any doubt about distal thrombosis, the backwashing technic of Shaw should be given every consideration, (8) either sympathectomy or sympathetic paralysis as herein described should be accomplished if arterial spasm seems to be a problem.

SUMMARY

Two cases of arterial embolism are herein presented.

Arterial embolism continues to be a serious problem with a high morbidity and mortality rate.

Auricular fibrillation, myocardial infarction with mural thrombosis and bacterial endocarditis are the leading causes.

The majority of emboli occur to limb arteries.

The most important consideration of these patients is early embolectomy.

Persistent arterial spasm and distal thrombosis are the major deterrents to success in the surgical therapy of this condition.

It is suggested that the *"backwash"* technique recently revived by Shaw be used more widely.

It is further suggested that continuous spinal anesthesia to interrupt sympathetic impulses, and yet cause little interference with muscular strength or cutaneous sensibility, is an easy and safe way to overcome persistent arterial spasm when sympathectomy is not done for one reason or another.

This may be accomplished by means of an indwelling small plastic catheter connected to a dilute Procaine solution. It is believed that the procedure may be a significant addition to our therapeutic armamentarium.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Edwards, W. S., and Lyons, C.: Traumatic arterial spasm and thrombosis, Ann. Surg. *140:* 318 (Sept.) 1954.
2. Kinmonth, J. B., Simeone, F. A., and Perlow, V.: Factors affecting diameter of larger arteries with particular reference to traumatic spasms, Surgery *23:* 452 (Sept.) 1949.
3. Linton, R. R.: Arterial embolism; simplified technique for removal of saddle embolus at bifurcation of aorta with report of successful case, Surg., Gynec. & Obst. *80:* 509 (May) 1945.
4. Martin, W. B., Laufman, H., and Tuell, S. W.: Rationale of therapy in acute vascular occlusions based upon micrometric observations, Ann. Surg. *129:* 476 (April) 1949.
5. Proffitt, J. N., and Elrod, P.: Arterial embolism from a symposium on peripheral vascular disease, J. Tennessee M. A. *45:* 352 (Sept.) 1952.
6. Richards, R. L.: Effects of peripheral arterial embolism, Quart. J. Med. *23:* 73 (Jan.) 1954.
7. Shaw, R. S.: More aggressive approach toward restoration of blood flow of acute arterial insufficiency, Surg., Gynec. & Obst. *103:* 279 (Sept.) 1956.
8. Warren, R., Linton, R. R., and Scannell, J. G.: Arterial embolism; recent progress, Ann. Surg. *140:* 311 (Sept.) 1954.

THROMBOSIS OF THE VENA CAVA TO THE LEVEL OF THE RENAL VEINS WITH RECOVERY

RICHARD A. DILLARD, M.D.,* CHAMP LYONS, M.D.,†
PAUL P. SALTER, JR., M.D.†

Birmingham

Thrombosis of the inferior vena cava and the renal veins with hemorrhagic infarction of the kidneys occurs as a complication of diarrhea of the newborn.[4, 1] In the adult thrombotic occlusion of the inferior vena cava, associated with ileofemoral thrombophlebitis, may progress to occlude the renal veins and result in death.[10, 5, 3, 8, 6]

The case of an adult male with this syndrome is presented who recovered following aspiration of a clot from the inferior vena cava at the level of the renal veins.

CASE REPORT

S. H. H.: This 59 year old white male farmer was admitted to the Birmingham Veterans Administration Hospital on Aug. 15, 1955, 15 days after the onset of spontaneous ileofemoral thrombophlebitis with pulmonary embolism, first treated elsewhere as pneumonia and later as infarction with heparin. On admission he complained only of painful swelling of the left leg. The lungs were clear to percussion and auscultation without friction rubs or rales. There was a normal sinus rhythm and no murmurs. The abdomen was soft with no tenderness or spasm. The liver was palpable but no other masses were found. There was obvious swelling of the entire left lower extremity to the inguinal ligament and over the buttock, with generalized tenderness more marked in the calf and medial aspect of the thigh. There was associated pitting edema over the entire left leg. The peripheral pulses were palpable and equal and there was no venous distention. The blood pressure was 130/70 in both arms.

The chest film showed a recent pulmonary infarct. The initial impression was ileofemoral thrombophlebitis and resolving right pulmonary infarction. The laboratory studies revealed a normal hemogram, a coagulation time of 8 minutes and a negative Kahn. The urinalysis was normal except for a trace of albumin and 9–14 white blood cells per high power field.

The patient was given intravenous heparin and placed on strict bed rest with the legs elevated. During the next 72 hours, the clotting time was checked every 6 hours by the Lee-White method and remained between 21 minutes and 45 minutes. The symptoms and signs subsided and he was placed on intramuscular concentrated sodium heparin. A friction rub developed over the right base posteriorly. The clinical improvement continued for 10 days, but he then developed a sudden occlusion of the arterial circulation of the right hand. The right radial pulse was diminished and the hand was hyperesthetic. The blood pressure was 130/80 in both arms. The electrocardiogram was normal. This improved in 48 hours.

During continuing heparin therapy, there developed signs and symptoms of ileofemoral thrombophlebitis is the opposite leg 25 days after admission. Intravenous heparin was again substituted for intramuscular therapy with improvement for 1 week. There then occurred abdominal pain, nausea and vomiting, with a blood pressure rise to 220/90. The electrocardiogram was normal apart from clinically apparent premature beats. The lungs

* From the Veterans Administration Hospital, Birmingham, Alabama.
† From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

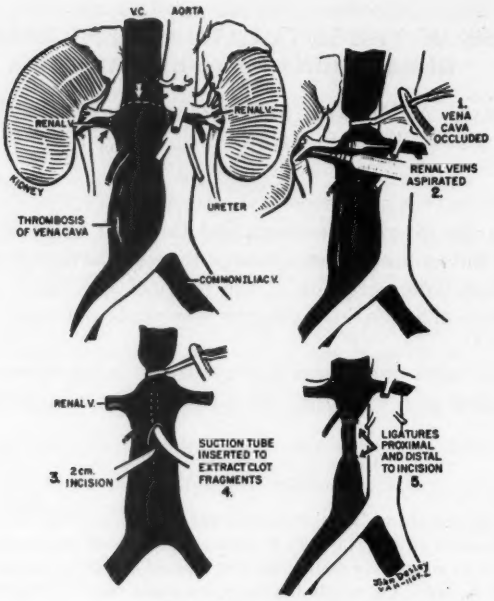


FIG. 1. Operative steps

were clear. There was diffuse lower abdominal tenderness with normal peristalsis. He had 3 bloody stools and innumerable red blood cells were present in the urine. There developed recurrent bilateral signs of ileofemoral phlebitis with absent arterial pulses below the femoral level. The clotting time was over 50 minutes and heparin was discontinued.

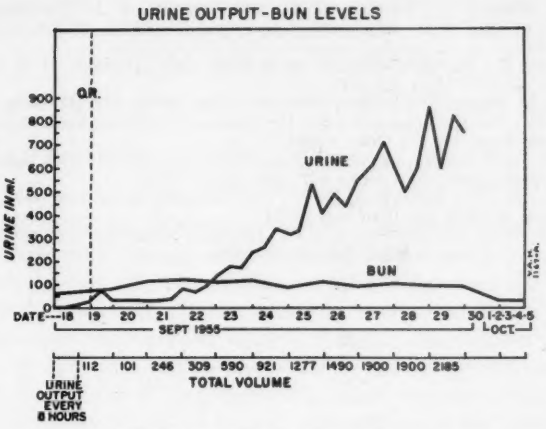
Forty-eight hours later the stools and urine were still bloody and oliguria was recognized. Apathy and extracellular fluid deficit were present with a blood urea nitrogen of 61 mg. per cent.

A clinical diagnosis of ascending thrombosis of the inferior vena cava with involvement of the renal veins seemed warranted. The poor prognosis of this complication, and the established failure of heparin above, lent support to the decision to attempt surgical removal of the clot.

A midline transperitoneal approach revealed no significant findings on abdominal exploration. Retroperitoneal examination disclosed an extensive fibroplastic reaction about the vena cava.

Palpation confirmed the presence of a firm clot extending from both iliac veins to within 1 cm. of the renal veins. Renal obstruction was thought to be present, for no blood could be seen eddying into the cava from the renal veins.

By means of an umbilical tape, the vena cava was occluded above the renal veins and blood was aspirated by needle from both renal veins (fig. 1). The vena cava was then opened through a 2 cm. longitudinal incision at the proximal end of the palpable clot. The clot partially extruded and was aspirated by placing a glass suction tip proximally. Much "currant jelly" clot was seen to pass through the tube. Only after suction had been completed above the level of the renal veins, was an active flow of blood obtained. No bleeding from the lower end occurred. The cava was then ligated below the renal veins. The right kidney was exposed and presented a bluish mottled appearance consistent with venous suffusion. There was 1 irregular bluish infarct about 1 cm. in diameter on the surface. No biopsy was done.



GRAPH I. Urine output blood urea nitrogen levels

Postoperatively the abdominal pain was relieved dramatically. Urine flow averaged 5 cc. per hour for the first 3 days after operation (see graph). Fluids and electrolytes were restricted in accordance with our program for anuric states.

Heparin was resumed 12 hours after operation. The legs were elevated and wrapped with elastic bandages. Late on the third postoperative day, urine flow began to increase. The electrolyte pattern was that of hypochloremic hyponatremic acidosis with maximal elevation of serum potassium to 5.48 mEq/l and blood urea nitrogen to 130 mg. per cent. The diuretic recovery phase was not remarkable. The convalescence was complicated by a severe diarrhea for 3 days, orchitis and epididymitis. He was discharged 8 weeks after admission with elastic bandages and no specific therapy. Six weeks after operation, the blood urea nitrogen was 24.5 mg. per cent and the urine contained red cells and albumin.

Phenolsulphonphthalein excretion was 5 per cent in 18 minutes and 57 per cent in 2 hours. Intravenous pyelograms revealed no definite pathologic changes, but dye excretion was delayed and visualization was poor. The patient was asymptomatic and physical examination revealed no abdominal tenderness or peripheral edema.

SUMMARY

A case of thrombotic occlusion of the inferior vena cava to the level of the renal veins with anuria for 24 hours prior to operation is reported. Recovery followed the aspiration of the clot and inferior vena cava ligation, although hemorrhagic infarction of the kidney occurred.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Campbell, M. F., and Matthews, W. F.: Renal thrombosis in infancy; report of 2 cases in male infants urologically examined and cured by nephrectomy at 13 and 33 days of age, J. Pediat. *20:* 604 (May) 1943.
2. De Bakey, M. D.: (Editorial) Problem of thrombo-embolism, Ann. Surg. *132:* 158 (July) 1950.
3. Derow, H. A., Schlesinger, M. J., and Savetz, H. A.: Chronic progressive occlusion of inferior vena cava and renal and portal veins with clinical picture of nephrotic syndrome; report of case with review of literature, Arch. Int. Med. *63:* 626 (April) 1939.

4. Feriozi, D., Rice, E. C., Burdick, W. F., and Troendle, F. J.: Thrombosis of inferior vena cava and renal veins with hemorrhagic renal infarction in infancy, J. Pediat. *38:* 235 (Feb.) 1951.
5. Lutembacher, R.: Thrombo-phlebite de la veine cave inferieure et de la veine porte, Presse Med. *43:* 1175 (July 24) 1935.
6. Madden, J. L.: Ligation of inferior vena cava, Ann. Surg. *142:* 200 (Aug.) 1954.
7. Myhre, J. H.: Venous gangrene with thrombosis of inferior vena cava: case report, A.M.A. Arch. Surg. *69:* 732 (Nov.) 1954.
8. Oaks, W. W., and Hawthorne, H. R.: Pseudoembolic phlebitis with ligation of inferior vena cava; case report, Ann. Surg. *127:* 1247 (June) 1948.
9. Shea, P. C., Jr., and Robertson, R. L.: Late sequelae of inferior vena cava ligation, Surg., Gynec. & Obst. *93:* 153 (Aug.) 1951.
10. Weber, F. P., Rast, H., and Wendt, L.: Thrombosis of inferior vena cava and prognosis of condition, M. Press. *196:* 262 (March 30) 1938.

MESENTERIC THROMBOSIS

S. Joseph Campbell, M.D., Thomas W. Mears, M.D.

Birmingham

INTRODUCTION

The surgical profession seldom encounters a greater challenge than that presented by mesenteric thrombosis. Its etiology remains obscure; its diagnosis too often is established only after the initial incision has been made, and in no situation does time become a more masterful enemy. Its many victories are cited in postmortem examination reports. Surgical intervention has been acclaimed the only successful therapeutic measure, and its value decreases, almost literally, with the passing of every hour. Therefore, we believe that any case of mesenteric thrombosis which is treated surgically and the patient recovers deserves reporting.

Etiology: While the etiology of this disease is, as yet, unknown, the following factors, are contributory:

1. Debilitating and degenerative diseases.
2. Inflammatory lesions of the abdomen.
3. Neoplastic lesions of the abdomen.
4. Mechanical factors.
5. Blood dyscrasias.

Mesenteric vascular occlusion may be arterial, venous or both. In arterial thrombosis, the superior mesenteric artery is almost always involved. Giamarino and Jaff[4] believe that this is due to the fact that the superior branch comes off the aorta earlier and also because it forms a more direct continuation of the abdominal aorta. Single or multiple hemorrhagic infarcts can be found, which may be due to emboli (from vegetations on the cardiac valves) or thrombi (due to arteriosclerosis or pressure from an abdominal tumor). A review of the literature indicates that the incidence of arterial and venous thrombosis is about equal.

However, of the 51 cases reported by Jenson and Smith,[14] arterial occlusion occurred more than twice as often as venous occlusion, the ratio being 29 to 12; the remaining 10 cases, which could not be catalogued as to type, presented clinical pictures indicating that they were mainly of the arterial type.

On the other hand, 60 cases presented by Whittaker and Pemberton[21] showed that only 19 were arterial, while 27 were venous and 14 were combined. Of the 19 arterial cases, 18 were due to obstruction of the superior mesenteric artery.

Uricchio, Calenda and Freedman[20] stated that in 1,142 cases reported by Bowen, the arteries were involved in 51.2 per cent, the veins in 43.8 per cent and both in 6 per cent.

Whittaker and Pemberton[21] revealed that 60 per cent of their cases were not

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

related to previous surgery, while 40 per cent were. They suggest that embolism is a more common cause of this disease than thrombosis. Uricchio, Calenda and Freedman[20] think that mycardial infarction and rheumatic heart disease with fibrillation are significantly associated with this disease. Jenson and Smith[14] stated that arteriosclerotic or rheumatic cardiovascular disease was present in 80 per cent of the arterial occlusion in their series. Their next most frequently noted finding was the fact that the patient had recently undergone abdominal surgery. In 19.5 per cent of their patients, an exploratory laparotomy had been done within 30 days before admission. Splenectomy for congenital hemolytic jaundice is perhaps the most often reported operation preceding mesenteric thrombosis, but Jenson and Smith[14] found this to be the case in only one instance in their series.

Venous obstruction is usually thrombotic and most frequently is caused by infectious processes of the abdominal organs. The muscular structures of the intestines remain viable longer in this type of occlusion. Experimentally, it has been proved that the small intestines may recover spontaneously from venous obstruction, provided the involvement does not exceed 6 inches. It is of interest that the terminal 4 inches of the ileum is not usually involved, since it is supplied by the ileocolic artery. The location of the occlusion and the number of occlusions determine the degree of permanent damage.

Symptoms and Diagnosis: There are no cardinal signs and symptoms which, if presented, would immediately signify a diagnosis of mesenteric thrombosis. The symptoms are not unlike those of intestinal obstruction, appendicitis, perforated peptic ulcer, acute cholecystitis, acute gastritis, food poisoning, simple enteritis, acute pancreatitis or volvulus of the bowel and intussusception. Therefore, unless this entity is constantly kept in mind, the correct diagnosis may be delayed until the patient has passed the operable stage. Some[14] believe that a diagnostic peritoneal aspiration is of value because sanguineous ascitic fluid is invariably found in mesenteric venous thrombosis. Jenson and Smith[14] found that this was true in 78 per cent of their cases. We have had no experience with this diagnostic procedure. Some[14] found that by inserting a polyethylene tube with multiple perforations, small amounts of bloody ascites may be noted when all other methods fail.

Donaldson and Stout[6] believe that a diagnosis can be made as to the type of obstruction; that is, whether it is arterial or venous. In the instance of arterial obstruction, it is even more imperative that early diagnosis and prompt treatment be rendered.

The following symptoms are usually found in venous obstruction:

1. Slow onset.
2. Colicky pain.
3. Abdomen tender to deep palpation.
4. Coffee ground vomitus.
5. Occult blood.
6. Intestinal fluid levels on roentgenogram absent.
7. White blood cell count not markedly elevated.

8. Engorged intestinal wall may, or may not, be palpable as a mass.
9. Temperature usually low.
10. Blood on glove on rectal examination.

Arterial obstruction differs from the above in these points:

1. Sudden onset.
2. Violent or extreme pain.
3. Rapid onset of gangrene, with corresponding clinical symptoms.

Arterial and venous conditions have the following points in common:

1. Tenderness.
2. Rigidity.
3. Distention.

It is of interest that this condition occurs twice as often in males as in females and, as previously stated, more often with advanced age. However, Whittaker and Pemberton[21] reported 1 case which occurred in a 13 year old female. Uricchio, Calenda and Freedman[20] found vomiting present almost invariably, but hematemesis occurred in only 1 patient. Tenderness and rigidity were not characteristic, and represented a late manifestation, appearing with the onset of peritonitis. This occurred in 10 of the 13 patients in their series. In 9 of their patients, distention was present, being localized at first and gradually becoming more generalized. In all of their patients, marked leukocytosis was present, ranging from 16,000 to 46,000 and averaged 25,000 per cu. mm. Jenson and Smith[14] noted leukocytosis and neutrophilia in over 88 per cent of their patients.

Rendich and Harrington[18] believed that roentgenologic diagnosis has too often been overlooked in arterial obstruction. In several patients they observed the large intestine distended over the left flexure (the area supplied by the superior mesenteric artery) with a very sudden cessation of this distention. In contrast to a mechanical obstruction, barium will pass through. No fluid levels are seen.

Treatment: In the surgical treatment, the following points should be borne in mind. Inspection of the affected area for viability will be the best index as to the amount of intestine to be removed. Douglas[8] suggested a needle prick through a vessel close to its intestinal attachment. Observation of the circulation here will be an excellent guide as to the point of resection. Should ⅓ to ½ of the intestine have to be removed, the patient may still survive. Following surgery, anticoagulants have been used by many, but we believe that they are of no significant value in the postoperative management of this disease. Jenson and Smith[14] found no difference in mortality whether anticoagulants were used or not, but they thought they were of value in preventing further extension of a thrombus after adequate removal of infarcted segments of bowel.

Prognosis: Without surgery, the mortality rate in mesenteric thrombosis approximates 100 per cent; with surgery, it approximates 30 per cent. The prognosis is brighter in venous than in arterial occlusion, but remains grave in any case. The mortality increases most rapidly with an increase in the loss of time between the period of onset and the time of surgery. We do not believe that early surgery can be advocated too strongly, as prompt surgical intervention often is the difference between life and death. We agree with those who believe that the results

of extensive laboratory studies should never be awaited, but that surgery should be initiated at the earliest possible moment. In operations performed on the first day after onset of clinical symptoms, the mortality approximates 25 per cent; while in patients operated upon on the fourth day, the mortality averages 83 per cent. Jenson and Smith[14] had no operative deaths and a mortality of 11.1 per cent in their cases of venous occlusion, with an 88.6 per cent mortality from superior mesenteric arterial occlusion.

SUMMARY AND CONCLUSIONS

Five cases of mesenteric thrombosis have been presented, 4 of the patients were able to return to work and maintained excellent health, even though massive bowel resection was performed. One patient died 2 days after surgery. Because of the location of the thrombus with gangrene of the entire bowel, resection was impossible.

A review of the literature suggests that cardiovascular disease and recent abdominal surgery are perhaps the two most frequently found conditions associated with this disease.

Early diagnosis and immediate surgery is emphasized as the only successful treatment. This entity should be constantly kept in mind when confronted with unexplained abdominal pain in all patients, but particularly so when dealing with those who are over 40 years of age.

REPORT OF CASES

Case 1. Mrs. I. H., a 68 year old white woman, was hospitalized on Aug. 22, 1955, with a history of cramping abdominal pain, nausea and vomiting of 4 days' duration. She had been on digitalis for 3 years for cardiac insufficiency. She said that her appendix had been removed some 25 or 30 years previously. An admitting diagnosis was made of intestinal obstruction, possibly due to carcinoma of the splenic flexure. A flat plate of the abdomen revealed fluid levels in the large bowel, which was distended over to the splenic flexure. There was no gas in the descending colon. Her white blood cell count was reported as 13,800 with 13.5 grams hemoglobin (88 per cent), with 11 lymphocytes, 12 monocytes and 77 neutrophils. She was carried to the operating room within a few hours after admission and an exploratory laparotomy revealed a massive intestinal gangrene, due to occlusion of the superior mesenteric artery. An occasional pulsation was felt along the superior mesenteric artery and it was decided not to resect the questionable viable intestine, because of the magnitude of the resection which would be required, and it was extremely doubtful that she could survive such an extensive procedure. Exploration of the superior mesenteric artery as carefully and systematically carried out, but no definite point of obstruction was found where an embolectomy could be carried out. Much to our surprise, exploration of the cecum revealed what appeared to be an acute, suppurative appendix. This diagnosis was confirmed on microscopic examination. The appendix was removed and the abdomen closed. She was given massive doses of antibiotics postoperatively and had an uneventful night; however, she became progressively weaker and died 2 days later. Postmortem examination revealed marked atherosclerosis, particularly involving the aorta, superior mesenteric arteries, splenic arteries and renal arteries. The superior mesenteric arteries were thrombosed and there was infarction of the small intestines with gangrene, and some changes in the cecum and ascending colon. There was a mild secondary hepatitis.

Case 2. A. O., 41 year old Negro man, was admitted to the hospital on April 9, 1952, in a moderate degree of shock and complaining of mild, generalized abdominal tenderness.

He had a normal bowel movement that day and had never had a previous similar episode. In 1951, an exploratory laparotomy had been done for partial small bowel obstruction, but otherwise he had been well until this hospital admission. On examination, the pulse was rapid and his blood pressure was 90/60. The heart was irregular and, in consultation, it was thought that this man might possibly have suffered a coronary occlusion. The abdomen was not distended and rectal examination was negative. The leukocyte count was normal. Twelve hours later, the abdomen was distended and exquisitely tender throughout. An electrocardiogram was reported as showing no evidence of coronary thrombosis. In spite of measures to combat his shock, he continued critically ill. It appeared as though he had an acute surgical condition within the abdomen, and the diagnosis of mesenteric thrombosis was entertained. The abdomen was explored and there was approximately 600 cc. of bloody transudate in the free abdominal cavity. The entire ileum and lower ⅓ of the jejunum was gangrenous. The gangrenous bowel was resected and a primary anastomosis between the jejunum and the proximal transverse colon was carried out. He recovered from his operation and, after a rather prolonged hospital course, returned to work. One year later he was doing well and maintaining his nutrition. He was having 3 to 4 bowel movements daily.

Case 3. Mr. G. A., 66 year old white man, was hospitalized on Aug. 22, 1951. Examination revealed an acutely ill man with a history of abdominal cramps for 24 hours prior to admission. His last bowel movement was 2 days previously, despite the fact that he had taken several laxatives, including epsom salts, without results. A flat plate of the abdomen showed multiple loops of distended bowel. He was immediately carried to the operating room and exploratory laparotomy with lysis of adhesions in the region of the ileocecal junction was performed. The obstruction was apparently relieved and he had an uneventful postoperative course for approximately 48 hours. Suddenly, he became markedly distended and was again explored. The exploration revealed gangrene of the small intestines with extensive thrombosis of the mesenteric vessels. Resection of approximately 8 feet of the small intestine was done, anastomosing the small intestine to the transverse colon. He made an uneventful recovery and was discharged from the hospital on Sept. 10, 1951. He enjoyed good health until several years later, at which time he developed pneumonia, which proved fatal.

Case 4. Mr. B., aged 65, was hospitalized because of mild abdominal pain at 10 p.m. Questioning revealed that he had eaten a tuna fish sandwich for lunch and shortly thereafter experienced severe abdominal cramps, nausea and vomiting. He took a large dose of Sal Hepatica that afternoon, which produced 2 large black stools. He did not seek medical advice until 8 p.m. His physician sent him to the hospital by ambulance, where an admitting diagnosis of acute gangrenous appendicitis was made. The abdomen was slightly distended and diffusely tender throughout, with maximum tenderness localized to the right of the umbilicus. There was a moderate degree of muscle spasm and rebound tenderness. Rectal examination was negative except for tenderness high in the right lower quadrant. No masses were palpable and no blood was evident on the examining glove. Routine laboratory studies revealed a white blood cell count of 18,250 with 88 per cent neutrophils and urinalysis to be within normal limits. A flat plate of the abdomen showed no gas under the diaphragm and only a few loops of distended small bowel. It was decided to operate. On entering the abdominal cavity, approximately 500 cc. of bloody fluid was encountered. A large area of gangrenous bowel involving the distal jejunum and ileum was found. The area of gangrenous bowel was well demarcated and there was an effusion of old blood into the mesentery, which extended up to the origin of the vessels. A resection of 61 inches of small bowel was performed, removing as much of the mesentery as possible. The continuity of the bowel was re-established with a side to side, isoperistaltic anastomosis. His convalescence was not noteworthy, except for the fact that a cervical lymph node abscess developed on the twelfth postoperative day. Two months following surgery, he returned to his job and has been in excellent health since that time.

Case 5. Mr. D. H. B., aged 59, was hospitalized on Aug. 15, 1947, suffering such excruciating pain that he was unable to give a history of his illness. His wife said that 20 hours pre-

viously he had begun having severe cramp-like abdominal pains about the umbilicus, of such severity that the opiates administered every 3 to 4 hours by his physician, gave only slight relief. He had experienced no nausea, diarrhea or melena. The physician advised us that the patient had been treated in the past year for upper abdominal distress and, on occasions, an irregular heart rate had been noted. Abdominal examination on admission revealed diffuse tenderness throughout, with marked rigidity. Leukocytes were 16,000 per cu. mm., with 94 per cent neutrophils on differential study. It was decided that he had an arterial type mesenteric thrombosis and abdominal exploration was elected. Upon entering the abdominal cavity, approximately 300 cc. of free bloody fluid was encountered in the peritoneal cavity. Beginning just proximal to the mid jejunum the bowel was gangrenous for approximately 8 feet. The lines of demarcation between normal and gangrenous bowel were sharply defined and there was extravasation of blood into the mesentery of the gangrenous bowel. A primary end to end anastomosis was done, after resecting the gangrenous bowel. He made a rapid recovery and his wound healed by primary intention. He was discharged from the hospital 10 days after admission. Five months later he developed homologous serum jaundice, which responded satisfactorily to medical measures. He has regained his weight and strength, his bowels are regular and he is entirely well. The cardiac rhythm has been restored by digitalis, administered in small daily doses.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Berger, S. S., and Blondis, R. T.: Mesenteric thrombosis, Am. J. Digest. Dis. & Nutrition *3:* 300 (July) 1936.
2. Blue, J. H., and Lafferty, C. R.: Mesenteric vascular occlusion, South. M. J. *33:* 968 (Sept.) 1940.
3. Brown, M. J.: Mesenteric venous occlusion; clinical entity, Am. J. Surg. *49:* 242 (Aug.) 1940.
4. Campbell, S. J.: Mesenteric thrombosis, massive resection of small bowel with recovery, J.A.M. Alabama *17:* 304 (March) 1948.
5. —— and Green, R. C.: Mesenteric thrombosis, massive small bowel resection with recovery, Mississippi Doctor *26:* 277 (March) 1949.
6. Donaldson, J. K., and Stout, B. F.: Mesenteric thrombosis, Am. J. Surg. *29:* 208 (Aug.) 1935.
7. —— and Sive, E. B.: Venous mesenteric occlusion, Surgery *6:* 80 (July) 1939.
8. Douglas, J.: Mesenteric vascular occlusion, Ann. Surg. *102:* 636 (Oct.) 1935.
9. Fallis, L. S.: Mesenteric thrombosis; operation and recovery, Am. J. Surg. *47:* 128 (Jan.) 1940.
10. Ficarra, B. J.: Mesenteric vascular occlusion, Am. J. Surg. *66:* 168 (Nov.) 1944.
11. Fox, P. F.: Mesenteric thrombosis with postoperative administration of dicumerol, Illinois M. J. *86:* 314 (Dec.) 1944.
12. Geis, A. F.: Mesenteric thrombosis; case with recovery, Am. J. Surg. *65:* 268 (Aug.) 1944.
13. Giamarino, H. J., and Jaff, S. A.: Mesenteric vascular occlusion, Arch. Surg. *45:* 647 (Oct.) 1942.
14. Jenson, C. B., and Smith, G. A.: Clinical study of 51 cases of mesenteric infarction, Surgery *40:* 930 (Nov.) 1956.
15. Laufman, H., and Scheinberg, S.: Arterial and venous mesenteric occlusion, Am. J. Surg. *58:* 84 (Oct.) 1942.
16. Luke, J. C.: Venous thrombosis, treatment with heparia, Lancet *1:* 552 (May 1) 1943.
17. Matthews, S. W.: Acute arterial mesenteric intravascular occlusion, Mil. Surgeon *80:* 223 (March) 1937.
18. Rendich, R. A., and Harrington, L. A.: Roentgenologic observation in mesenteric thrombosis, Am. J. Roentgenol. *52:* 317 (Sept.) 1944.
19. Scott, R. A. M., and Lissimore, N.: Mesenteric thrombosis in lymphatic leukemia treated with dicumerol, Lancet *2:* 405 (Sept.) 1944.
20. Uricchio, J. F., Caldenda, D. G., and Freedman, D.: Mesenteric vascular occlusion, Ann. Surg. *139:* 206 (Feb.) 1954.
21. Whittaker, L. D., and Pemberton, J. deJ.: Mesenteric vascular occlusion, J.A.M.A. *111:* 21 (July 2) 1938.

SPLENECTOMY, THROMBOCYTOSIS, AND VENOUS THROMBOSIS

PAUL P. SALTER, JR.*, M.D., EUGENE C. SHERLOCK, B.S.†

Birmingham

The literature of the past few years contains many references concerning both abdominal and peripheral thrombosis following splenectomy. With the increasing number of splenectomies for primary splenic pathology, for trauma and for completeness of cancer surgery, it seemed wise to review our experience with 101 consecutive splenectomies over the past 6 years with special reference to thrombocytosis and vein thrombosis.

Delatour,[4] in 1895, reported portal vein thrombosis and death as a complication of splenectomy for "simple splenic hypertrophy." The earlier literature[1, 3, 7, 19] stressed splenic and portal vein thrombosis as a complication of splenectomy performed for congestive splenomegaly, Banti's disease, and Wilkins[27] listed as contributing factors: (1) stasis incident to splenomegaly, (2) dilatation and tortuosity of the splenic vein, (3) operative trauma, (4) ligation of the splenic vein, and (5) infection. Davis and Sharpe[3] reported a 5.7 per cent incidence of portal and mesenteric thrombosis postsplenectomy in 193 patients with Banti's disease.

Simonds[21] attached much significance to alteration in blood composition, namely the occurrence of thrombocytosis, as a predisposing factor to thrombosis. This increase in platelets is a fairly constant physiologic sequel of splenectomy. It begins almost immediately postsplenectomy and may reach a peak of 1 to 2 million by the tenth to twelfth postoperative day, and may persist for several years.[10, 22, 25] Rosenthal[19] and Howell Evans[7] studied preoperative and postoperative platelet counts in Banti's disease and associated a high incidence of portal thrombosis with patients who present high normal preoperative counts and marked thrombocytosis postsplenectomy (thrombocythemia). Bryce[1] agreed with the dangers of splenectomy in the thrombocythemic group, but reported a case with marked thrombocytopenia pre and postsplenectomy who developed portal thrombosis leading to death. W. J. Mayo[13] stated that 10 per cent of postsplenectomy deaths were due to thrombosis.

One gets the impression from various writers that splenic and portal vein thrombosis is a frequent complication of splenectomy in general. Kelly[11], in 1929, citing 2 cases of marked thrombocytosis postsplenectomy, stated:

"We believe that postoperative thrombosis is generally, but not invariably, associated with a thrombocytosis. Splenectomy is a safe operation if the thrombocyte count is below 200,000, but is a dangerous one when the thrombocyte count approaches 1,000,000. The patient then runs the risk of thrombosis in the portal and mesenteric veins."

* From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.
† Senior Medical Student, Medical College of Alabama, Birmingham, Alabama.

MATERIAL

This report includes all splenectomies performed at the University Hospital and Veterans Administration Hospital from Jan. 1, 1950, through May 30, 1956. The youngest patient was 1 day old and the oldest, 68 years of age. In all, 101 splenectomies were performed; of these 61 were males and 40 females.

INDICATION FOR SPLENECTOMY

The lesions treated included primary splenic pathology, traumatic rupture and incidental splenectomy. Twenty-five splenectomies were performed for primary splenic pathology, 41 for traumatic rupture and 35 incidental to other surgery. Splenectomies for traumatic rupture occurred most frequently in the third and fourth decades, whereas the peak incidence of splenectomy for primary splenic pathology and completeness of cancer surgery was in the fifth and sixth decades.

TABLE 1

Indications for splenectomy

Disease	No. of Patients	Percentage
Traumatic rupture	41	40.6
Penetrating	20	
Blunt	21	
Primary splenic pathology	25	24.7
Idiopathic thrombocytopenia	9	
Portal hypertension with or without hypersplenism	8	
Congenital hemolytic anemia	2	
Sickle cell anemia	2	
Secondary thrombocytopenia	2	
Splenic cyst	1	
Unknown	1	
Incidental to other surgery	35	35.7

TABLE 2

Postoperative platelet counts

Platelets	Disease	No. of Patients	Percentage
Below 500,000		30	47.6
	Splenic pathology	9	
	Trauma	9	
	Incidental	12	
500,00 to 1,000,000		25	39.7
	Splenic pathology	11	
	Trauma	9	
	Incidental	5	
More than 1,000,000		8	12.7
	Splenic pathology	2	
	Trauma	4	
	Incidental	2	

TABLE 3

Anticoagulant therapy

Disease	Platelets	Signs of Venous Thrombosis	Drug
Lacerated spleen	4,000,000	None	Heparin
Primary splenic pathology	730,000	None	Heparin
Lacerated spleen	1,873,000	None	Heparin
Lacerated spleen	479,920	Pulmonary embolism	Dicumarol
Gastric carcinoma	Not done	Leg signs	Heparin

TABLE 4

Deaths

	Disease	Autopsy	Cause of Death
B. B.	Multiple fractured ribs, head injury, lacerated spleen	No	Head injury, pneumonitis
H. C.	Multiple rib fractures, lacerated spleen, fractured pelvis	No	Irreversible shock at surgery
W. W.	Gunshot spleen, stomach, colon, portal vein	No	Exsanguination at surgery
R. Y.	Splenomegaly with thrombocytopenia secondary to toxic hepatitis	No	Shock eighth postoperative day—cause undetermined
G. L.	Splenomegaly with thrombocytopenia, cirrhosis, portal hypertension	No	Exsanguination at surgery
C. J.	Stab wound of spleen, stomach, liver, subdiaphragmatic abscess	Yes	Peritonitis, 20th postoperative day
T. W.	Stab wound of spleen, subphrenic abscess	Yes	Peritonitis 18th postoperative day
B. K.	Congenital diaphragmatic hernia	Yes	At operation
H. B.	Multiple rib fractures, hemothorax, laceration of spleen	Yes	Flail chest, bilateral hemothorax
J. Y.	Duodenal ulcer, subtotal gastrectomy, subphrenic abscess left, peritonitis, evisceration	Yes	Peritonitis
H. D.	Stab wound of spleen, liver, celiac axis	Yes	Exsanguination
E. S.	Hiatal hernia, splenectomy—major blood reaction	Yes	Renal failure

Postoperatively platelet counts were obtained on all charity patients and careful examination for evidence of peripheral or portal thrombosis was routine. Postoperative platelet counts were obtained on 63 (62.4 per cent) patients. These counts were begun on the third postoperative day and done every third day thereafter for a total of four counts.

Early in the series anticoagulant therapy (heparin and/or dicumarol) was given prophylactically to those patients with platelet counts exceeding 1,000,000 by the tenth postoperative day or to those patients with clinical evidence of peripheral thrombosis.

The dosage of heparin varied, but was usually administered intravenously 100 mg. every 6 hours for a 10 day period. The ideal clotting time which was sought was 20 minutes. Dicumarol was given to lower and maintain the prothrombin time at 20–30 per cent of normal. Of 5 patients who received anticoagulant therapy, 3 were given anticoagulants prophylactically because of elevated platelet counts and 2 because of clinical evidence of peripheral thrombosis and/or pulmonary embolism. Later in the series 5 patients were found to have had platelet counts exceeding 1,000,000, but no specific treatment was given and none developed clinical venous thrombosis. A third patient in the series, who had a splenectomy for idiopathic thrombocytopenic purpura, developed peripheral venous thrombosis on the ninth postoperative day. Initial platelet count was 39,900 and the highest count was 889,280. No specific therapy was given.

DEATHS

There were no deaths in the patients who developed evidence of venous thrombosis or in those who developed a thrombocytosis exceeding 1,000,000.

Twelve deaths occurred during the postsplenectomy hospitalization. Seven (62.5 per cent) autopsies were obtained and in none of these was there evidence of portal vein thrombosis. Two deaths were associated with primary splenic pathology, 3 with splenectomy incidental to other surgery and 7 with splenic injury.

Three of the autopsied patients had splenic vein thrombosis without extension into the portal vein.

DISCUSSION

Three factors have been accepted as fundamental as to the causation of thrombosis in general.[21] These are: (1) alteration in blood composition, chiefly thrombocytosis, (2) slowing of the blood flow or stasis, and (3) injury to the lining of the vessels. Early reports associated venous thrombosis and splenectomy for congestive splenomegaly. They offered no means of preventing or treating the stasis or thrombocytosis, but did warn against splenectomy in patients with normal to high preoperative platelet counts because of the almost uniform occurrence of portal thrombosis and death.[1, 3, 7, 19] More recent reports have associated portal thrombosis following splenectomy for traumatic rupture as well as for completeness of cancer surgery.[8, 17] The latter, however, have the added incidence of spontaneous venous thrombosis associated with carcinoma.[2, 9, 23] Eliason and Stevens[6] reported no deaths from portal thrombosis in 63 splenectomies performed for primary splenic pathology.

The fear of thrombocytosis and venous thrombosis postsplenectomy stimulated many studies in their prevention and treatment. In 1929, Walton[26] advised citrates and early limb movements in patients with normal preoperative platelet counts. Moore[14] several years later (1936) attempted to lower the preoperative platelet count with bone marrow and splenic irradiation. The conversion of his patients from the thrombocytemic to the thrombocytopenic group was successful,

but the patients died of postoperative portal thrombosis. Diamond[5] also suggested deep roentgen therapy to the chest as a means of reducing the platelet count. Later Murray and MacKenzie[15] demonstrated experimentally in dogs that heparin could be of great value in preventing portal thrombosis after splenectomy. Clinically they used heparin prophylactically in patients without evidence of hematoma or venous thrombosis.

With these many reports of vein thrombosis following splenectomy and with the general acceptance of anticoagulant therapy to prevent postoperative thrombosis, we initially gave all patients anticoagulants prophylactically if the platelet count exceeded 1,000,000 by the tenth postoperative day.[16] Three patients were treated thus and none developed clinical evidence of venous thrombosis. It was the opinion of the staff, however, that our incidence of venous thrombosis was no higher following splenectomy than any other major surgical procedure. Stimulated by communications from Wintrobe[28] and Rhinelander,[18] both of whom did not use anticoagulants prophylactically, we reviewed the splenectomies herein reported.

Three (3%) of the 101 splenectomy patients developed evidence of peripheral thrombosis and/or pulmonary infarction. None of these had a platelet count exceeding 1,000,000 nor was there evidence of portal thrombosis. This is in accord with Rhinelander[18] who reported 410 splenectomies for blood dyscrasias performed at the New England Medical Center. Only 2 patients developed evidence of venous thrombosis postsplenectomy. Both patients had splenectomy for chronic idiopathic thrombocytopenic purpura and in spite of a platelet count below 100,000 developed spontaneous thrombosis. Several patients developed evidence of pulmonary infarction, but the incidence was no higher than in their general group of major surgical procedures. Martin and Cooper,[12] in a review of the literature stated: "Pulmonary and cerebral embolism, since it follows peripheral thrombosis, is not increased in splenectomy."

Terry, Self, and Howard[24] recently reported 102 splenectomies for trauma. Only one patient developed phlebitis as a postoperative complication. There was no portal thrombosis in the autopsied patients.

With the lack of any correlation between splenectomy, thrombocytosis, and thrombosis, in our series and in those of Rhinelander[18] and Terry,[24] we have stopped the prophylactic use of anticoagulants. Since the initiation of this policy 5 patients have developed platelet counts exceeding 1,000,000 but none have evidenced either peripheral or portal thrombosis. Patients with leg signs or evidence of pulmonary infarction have received standard therapy. Four patients in our series have had marked and prolonged unexplained febrile courses. Terry[24] recorded 2 such cases. These patients are probably examples of the modern day splenic and portal thromboses that fail to produce their full effect in an antibiotic protected liver and otherwise well supported patient.

CONCLUSION

In 101 splenectomies there was no increase in venous thrombosis and no incidence of portal thrombosis.

No true correlation was found between splenectomy, thrombocytosis, and venous thrombosis.

Prophylactic anticoagulants are no longer used postsplenectomy.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Bryce, A. G.: Splenectomy and thrombosis, Lancet *2:* 1423, 1932.
2. Cooper, T., and Barker, N. W.: Recurrent venous thrombosis; early complication of obscure visceral carcinoma, Minnesota Med. *27:* 31 (Jan.) 1944.
3. Davis, H. H., and Sharpe, J. C.: Splenic vein thrombosis following splenectomy, Surg., Gynec. & Obst. *67:* 678 (Nov.) 1938.
4. Delatour, H. B.: Thrombosis of mesenteric veins as a cause of death after splenectomy, Ann. Surg. *21:* 24 (Jan.) 1895.
5. Diamond, L. K.: Indication for splenectomy in childhood: results in 52 operative cases, Am. J. Surg. *39:* 400 (Feb.) 1938.
6. Eliason, E. L., and Stevens, L. W.: Surgery of spleen in blood dyscrasias, Surgery *13:* 177 (Feb.) 1943.
7. Evans, W. H.: Blood platelets in splenic anemia, Lancet *1:* 277 (Feb.) 1929.
8. Gius, J. M., McGovern, J. D., and McMurray, W. R., Jr.: Portal vein thrombosis following removal of ruptured spleen, Ann. Surg. *121:* 100 (Jan.) 1945.
9. Hubay, C. A., and Holden, W. D.: Venous thrombosis, necrosis and neoplasia, Surg., Gynec. & Obst. *98:* 309 (March) 1954.
10. Hitzrot, J. M.: Splenectomy on normal individuals, Ann. Surg. *67:* 540, 1918.
11. Kelly, R. E.: Symposium; discussion on indication for and results of splenectomy, Proc. Roy. Soc. Med. *22:* 1514, 1929.
12. Martin, J. D., Jr., and Cooper, M. N.: Complications of splenectomy, South. Surgeon *16:* 1047 (Nov.) 1950.
13. Mayo, W.: Collection of papers of Mayo Clinic, *17:* 625, 1925.
14. Moore, S. W.: Portal thrombosis following splenectomy for splenic anemia, Surg., Gynec. & Obst. *63:* 382 (Sept.) 1936.
15. Murray, G., and MacKenzie, R.: Effect of heparin on portal thrombosis; its use in mesenteric thrombosis and following splenectomy, Canad. M. A. J. *41:* 38 (July) 1939.
16. Norcross, J. W.: Splenectomy; its indications and complications, S. Clin. North America *24:* 583 (June) 1944.
17. Quan, S., and Castleman, B.: Splenic-vein thrombosis following transthoracic gastrectomy and incidental splenectomy, New England J. Med. *240:* 835 (May 26) 1949.
18. Rhinelander, H. F.: New England Medical Center, (Personal communication).
19. Rosenthal, N.: Clinical and hematologic studies on Banti's disease, J.A.M.A. *84:* 1887 (June 20) 1925.
20. Rousselot, L. M.: Present concepts in surgery of spleen, S. Clin. North America *29:* 369 (April) 1949.
21. Simonds, J. P.: Chronic occlusion of portal vein, Arch. Surg. *33:* 397 (Sept.) 1936.
22. Singer, K., Miller, E. B., and Dameshek, W.: Hematologic changes following splenectomy in man with particular reference to target cells, hemolytic index and lysolecithin, Am. J. Med. Sc. *202:* 171 (Aug.) 1941.
23. Sproul, E. E.: Carcinoma and venous thrombosis frequency of association of carcinoma in body or tail of pancreas with multiple venous thrombosis, Am. J. Cancer *34:* 566 (Dec.) 1938.
24. Terry, J. H.: Injuries of spleen, Surgery *40:* 615 (Sept.) 1956.
25. Wollstein, M., and Kreidel, K. V.: Blood picture after splenectomy in children, Am. J. Dis. Child. *51:* 765 (April) 1936.
26. Walton, A. J.: Symposium; discussion on indications for and results of splenectomy, Proc. Roy. Soc. Med. *22:* 1496, 1929.
27. Wilkins, J. A.: Embolism and thrombosis in portal system, Collection of papers from Mayo Clinic *15:* 736, 1923.
28. Wintrobe, M. M.: Stuart Graves lecture, University of Alabama, 1956.

EARLY MANAGEMENT OF THE PLEURAL SPACE FOLLOWING PARTIAL PULMONARY RESECTION

REX PERKINS, M.D.

Birmingham

A cardinal principle in the postoperative management of partial pulmonary resection patients is that the remaining portions of the lung should be totally expanded. Attainment of this goal requires that attention be directed toward (1) maintenance of healthy pulmonary parenchyma, (2) maintenance of an unobstructed tracheobronchial tree, and (3) total approximation of visceral to parietal pleura. In practice, these objectives are pursued (1) by the use of antibiotics and attention to fluid and electrolyte balance; (2) by means of early ambulation, elimination of tight dressings, relief of pain, coughing, aerosol medications, tracheobronchial catheter aspiration, and bronchoscopy; and (3) by mechanical removal of the contents of the pleural space by needle, tube, or open thoracotomy. This report is restricted to a consideration of certain concepts and details of the latter maneuver, i.e., evacuation of abnormal pleural space materials in the immediate postoperative period. It is helpful to approach the problem by classifying these materials as solid, liquid, or gas.

SOLID MATERIAL

The solid material will be clotted blood, accumulated from multiple oozing points or actual bleeding vessels on raw parietal, mediastinal, or pulmonary surfaces. The frequency and extent of postoperative clotted hemothorax can be minimized by certain maneuvers, such as (1) treatment of abnormal bleeding tendencies; (2) careful hemostasis before closure, including the use of artificial hemostatic agents such as gelfoam on particularly troublesome areas; (3) attention to known treacherous oozing areas, such as divided adhesions, bone ends, injured intercostal vessels, and raw pulmonary surfaces; (4) careful chest wall closure; (5) leaving 200–300 cc. of saline solution in the chest during closure so that clotting of the oozed blood will not occur during the crucial hour from the onset of closure to the time when the patient is turned and adequate tube drainage begins; (6) removing oozed blood quickly before it clots by efficient use of tubes, preferably with suction; (7) securing prompt expansion of the lung with obliteration of the pleural space. This latter maneuver is most important, as the tamponing effect of the expanded lung against the chest wall is the best deterrent to oozing from either. Once a clotted hemothorax is present however, the guiding principles are that there is usually no great urgency to remove the clot quickly unless it is very large, and that the clot should not be allowed to become infected. There are three methods of dealing with the clot, viz: leave it alone, dissolve it and remove it with a needle, or remove it by open thoracotomy.

In many instances a smaller clot, if uninfected, may be treated conservatively,

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

depending upon the strong resorptive ability of the pleura to deal with it. Varying amounts of the clot may undergo spontaneous liquefaction and thus may be removed with a needle, using utmost sterile precautions and concomitant injection of antibiotics following the tap. Clot resorption takes several weeks or months, but may be the advisable course to follow when the clot is small, when it is located in an awkward area such as between the lung and mediastinum, or when vigorous management of it would be unwise for any reason.

In many instances active efforts should be made to remove the clot for these reasons: (1) it may be actually or potentially infected; (2) it may be large enough to be embarrassing ventilation or to be causing mediastinal displacement; (3) it may be preventing pulmonary expansion in an undesirable manner; and (4) the ultimate pleural peel, even if marked resorption occurs, may be sufficient to markedly hamper rib motion, diaphragmatic, and lung expansion, and thus by destroying pulmonary function will mitigate much of the value of the original operation. In removing large or infected clots, much success can be attained by the use of the enzymatic debridement agents, streptokinase and streptodornase. Streptokinase activates the substrate plasminogen into plasmin, which liquifies the clot. Streptodornase is desoxyribronuclease, which liquifies exudative material, a large part of which is desoxyribronucleoprotein contributed by leukocytes and inflammatory debris. These agents, along with antibiotics, are introduced by needle, and the liquified material is removed 6 to 12 hours later by needle. If this course of treatment is elected, it must be pursued vigorously and the thoracentesis must be done carefully and on schedule. The liquified material has a characteristically muddy appearance, but this does not necessarily indicate that it is grossly purulent, as bacterial examination will often show. With this technic, very large clotted hemothoraces may be successfully managed. In addition, unless the clot is very grossly infected, this method of securing clot evacuation and lung expansion will usually prevent a frank empyema, or at least will abort its size and severity and make drainage a lesser maneuver. Certain drawbacks to the use of these agents should be mentioned: (1) a febrile reaction occurs, at least with initial injections; (2) actual severe anaphylactic reactions may occur;[2] (3) repeated thoracentesis carry increased risk of introduction of organisms, needle laceration of the lung, and injection of the enzymes into tissues; and (4) fibrin sealed alveolar leaks, such as occur after segmental resections or trauma, may reopen and create a pneumothorax problem. A healthy, properly sutured bronchial stump, however, should not leak after contact with the enzymes.[1]

A rubber drainage tube should not be inserted for treatment of clotted hemothorax, since the clot will not come out through the tube any better than through a needle, and because the clot will soon become infected by organisms passing in along the tube tract. If a clot forms near to the site where a tube is already in place anyway (to handle air or fluid), and especially if the clot is already infected, the tube may be used to give enzymatic debridement therapy as described above. It is likely however, that unless the clot can thus be quickly removed and the tube soon withdrawn, some terminal drainage of a small empyema pocket will be necessary. If a clot forms some distance from an already inserted tube, it

FIG. 1. Roentgenogram of a patient who had left apicoposterior segmental resection 8 days previously. Acute postoperative fibrinolytic reaction caused massive clotted hemothorax with resultant persistent fluid drainage, air leak, and unexpanded lung.

is best to remove the tube in routine fashion and treat the clot as a separate entity.

Formal thoracotomy for removal of clotted blood is sometimes necessary either in the early postoperative course or, more often, as a delayed procedure. Early postoperative surgical evacuation of the clot may be required when (1) there is progressive enlargement of the clot from uncontrolled oozing not adequately handled by tubes, (2) the clot is large enough to embarrass respiration or cause mediastinal shift, or (3) there is a concomitant air leak, requiring persistent intubation with liklihood of early infection of the clot (figs. 1, 2). Formal thoracotomy should include removal of the clotted material, attention to hemostasis, lysis and decortication of the lung, lavage of the pleural space, repair of any air leaks, and prompt total re-expansion of the lung, maintained by tube suction.

LIQUID MATERIAL

Several types of liquid material may accumulate in the pleural space in various circumstances, including serous transudate, chyle, and various inflammatory exudates. For our purpose in considering only the immediate post-thoracotomy situation however, we will discuss only the routinely accumulating serosanguinous fluid which oozes from raw and traumatized surfaces. This fluid may vary in quantity from practically none to several liters, depending upon such factors as (1) type of operation; (2) duration of surgery (3) presence or absence of infection; (4) adequacy of hemostasis; (5) the amount of intrathoracic trauma and the

FIG. 2. Roentgenogram of same patient as in figure 1, 2 months later. Tube suction, needle aspirations and use of streptokinase-streptodornase were unavailing. Repeat thoracotomy was done with evacuation of clot, suture of air leaks, decortication of lung, thoracoplasty, and further tube-suction drainage, with good result.

extent of raw surface; and (6) the rapidity and adequacy of obliteration of the pleural space; and (7) the presence of a postoperative oozing tendency, such as follows a mismatched transfusion. The duration of this fluid accumulation is usually about 72 hours, but again this varies with the above factors. The best deterrent to both quantity and duration of serosanguinous drainage is total pulmonary expansion with obliteration of the pleural space. This fluid may have a hematocrit of 20 vol. per cent or higher and represents a significant depletion of hemoglobin and blood volume which frequently requires replacement by transfusion.

The fluid may be mechanically removed by means of the needle, the tube or open thoracotomy. Needle aspiration of sanguinous fluid postoperatively may be employed when two criteria are met: (1) fluid production is expected to be minimal, and (2) the visceral pleura is intact and there will be no intrapleural air accumulation. Thus the method is applicable to simple exploration, vagotomy, certain mediastinal operations, some cardiovascular procedures, and even to some cases of pulmonary biopsy or lobectomy if there is no air leak. In these patients the chest may be closed primarily without drainage and from 1 to 3 thoracenteses usually suffice to remove the fluid. Thoracentesis is also of use in removing fluid accumulations which sometimes develop after extubation. Except where the volume of fluid is expected to be quite minimal, it is preferable to remove it continuously as it is produced in order to prevent loculation, clotting,

infection, and inadequate lung expansion. This continuous drainage of the fluid is best accomplished by indwelling tubes with suction. Certain technical aspects of the use of thoracic tubes and suction setups are quite important and are discussed fully below. Whereas it is quite true that fluid can be adequately removed in most instances by simply connecting the chest tubes to water trap bottles, in the author's opinion the concimitant use of continuous suction is immensely desirable in all patients. Other theoretic considerations being equal, suction can do no harm. Without suction, fluid removal is a passive intermittent process dependent upon patient-produced episodes of positive intrapleural pressure to force the fluid out of the tube. Gravity comes into play of course, but is imperfect and may even work against efficient drainage since patient position is frequently such that the fluid has to go over one or more "hills" in the tubing to get to the bottle. Likewise, the siphon effect is usually negated by mechanical factors such as the presence of air in portions of the tubing. Indeed, the patient frequently has to build up an intrapleural pressure of 20 cm. H_2O or more, as during coughing, to cause egress of chest fluid, for example, when the tube is led uphill over a pillow before descending to the bottle, or when there is a fluid-filled dependent loop in the tubing. These are details, of course, but adequate postoperative care is a matter of attention to details. In a subject admittedly difficult to document, it has been the author's impression that the use of continuous suction is of definite value in terms of efficient removal of the fluid, the prevention of loculation, the early uniform, and complete expansion of the lung, the reduction of pleural complications, and the reduction of total patient morbidity.

Thoracotomy for the removal of fluid should not be necessary except in those rare instances when there might be uncontrolled postoperative bleeding and a re-exploration required for its control. In all other instances fluid can be removed by needle or tube.

GAS

The gaseous material will be air, which accumulates by leakage from the lung or some portion of the tracheobronchial tree. Certain other sources, such as entrance from the esophagus, from the outside through a chest tube tract, or from technical accidents referable to gross mismanagement of a tube-suction set up, may occur but should be easily recognized. Also some air may be left in the pleural space at operation. In general, though, the pleural air represents bronchopulmonary leak arising as a result of trauma to the lung or bronchi. Lacerations and perforations of the parenchyma, leaking suture lines, raw surfaces from division of adhesions, leaking needle holes, raw surfaces after lobectomy or segmental resection, and actual bronchial stump leakage are some of the sources of intrapleural air postoperatively.

The leakage of air into the pleural space is a much greater problem than is the accumulation of fluid because the total amount of fluid is usually limited to a few hundred cc. over a course of several days, whereas the air leak may be very rapid and very persistent, with a volume which may be measured in terms of hundreds of cc. per minute. Thus the pneumothorax may be quite extensive and tension

pneumothorax may develop. A pleural air space mitigates against cessation of fluid ooze and against cessation of the air leak itself. The best method of sealing air leaks from the lung is to keep the lung expanded and thus plaster the raw leaking surface against some other surface. Latent infection tends to progress in the presence of a persistent pleural space, whereas obliteration of the space usually aborts infection. The rapidity with which infection develops in a pleural space fed by an air leak depends to a great extent upon the site in the bronchial tree from which the leak is occurring. When there are only persistent alveolar leaks, severe infection may not occur for several weeks, whereas if the leak is from a main bronchial stump, irreversible contamination may occur in a matter of hours. It is of great importance then to forsee and prevent postoperative pneumothorax. There are four methods of dealing with postoperative intrapleural air accumulations: (1) leave it alone; (2) thoracentesis; (3) thoracotomy; and (4) continuous tube drainage, with or without suction. The first three methods have limited usefulness and will be discussed first.

Inasmuch as the ability of the pleura to absorb air is very great, it may be wise to not disturb pleural air provided certain conditions are present, viz: (1) the air pocket should be small; (2) there should be no continuing air leak into it; (3) there should be very little associated fluid; and (4) the danger of infection should be minimal. Two examples may be given: (1) a 10 per cent pneumothorax exists due to air remaining in the chest after closure of a simple exploratory thoracotomy; (2) following segmental resection after removal of chest tubes there develops, due to a temporary slight alveolar leak, a small apical air pocket which does not enlarge on close roentgenologic following, which has no significant associated fluid, and in which the patient is afebrile. Strict rules for these situations cannot be formulated, and decisions in individual cases must be on the basis of the existing circumstances and the surgeon's experience.

Thoracentesis for the removal of air following elective surgery is sometimes necessary when an unexpected air space develops. Anticipated pneumothorax should, of course, have been originally treated by intubation, but on occasion air accumulations occur which were not forseen and provided for. Following extubation, for example, of a segmental resection, air leak may develop with resultant "pull-away" of the lung and pneumothorax of considerable size. In addition to removing the air, thoracentesis provides information regarding future management of the space. Thus the intrapleural pressure can be measured by manometer while the needle is in the space, and if a negative pressure cannot be maintained following removal of the air, there is an active air leak which should be treated by tube and suction. The same is true if an unlimited amount of air can be removed during the tap, or if roentgenologic follow-up shows recurrence or progression of the space. Technically, a needle can be inserted in any portion of any interspace. Thus the first interspace posteriorly can be tapped for apical air pockets, and the chest wall under the scapula can also be entered with suitable mobilization of the scapula and angulation of the needle. One complication of thoracentesis, which is sufficiently common to warrant special caution, is laceration of the lung by the sharp needle point. This accident produces a new air

leak, often of some magnitude, which may make the situation worse than it was previously and may even lead to total lung collapse. The careful use of a short beveled needle is valuable in this regard, but the best maneuver is the use of a short piece of polyethylene tubing. Following insertion of the needle, the tubing is threaded through it into the space, and the needle is withdrawn over the tubing. The air is then aspirated, and as the lung expands against the soft tubing it is not injured.

Thoracotomy is sometimes necessary to eliminate the source of a postoperative air leak which is of sufficient magnitude and persistence that it threatens not to seal spontaneously, to maintain a pneumothorax, and to lead to an empyema. Reoperation of a persistent postoperative bronchopleural or bronchiolo-pleural fistula should be carried out with certain considerations in mind; (1) an adequate trial of nonoperative treatment by intubation with suction has been unavailing, or the leak is of such magnitude as to indicate a large fistula which will not close on this regime; (2) gross infection is not yet present; and (3) effective obliteration of the pleural space after the reoperation should be assured, including the use of thorocoplasty if necessary (fig. 2).

Following partial pulmonary resections, the use of rubber tubes to remove air (and liquid) is standard procedure and is the safest and most efficient method. Suction is not necessary to achieve lung expansion when there is no intrathoracic air leak, as for example, following a lower lobectomy in which a complete major fissure was present. The first few episodes of positive intrapleural pressure, as with coughing or straining, will push most of the air out of the tube. It is the author's belief, however, that suction should be applied in all patients for these reasons: (1) cases of partial pulmonary resection in which there is no air leak will be in the minority, and it is not always possible to predict with certainty that there will not be an air leak: (2) the pleural space that must be obliterated by adjustments of the diaphragm, lung, mediastinum, and costal cage, as after a lower lobectomy, is sometimes fairly large; these adjustments seem to occur more readily under the stimulus of constant externally applied negative pressure; (3) with nonsuction drainage, total expansion is frequently delayed, requiring hours or days to occur; it is thought that *rapid*, maintained, total expansion is highly desirable; (4) controlled suction can do no harm.

There is widespread agreement that suction is necessary in the presence of an active air leak. Here there is a dynamic situation of a constant accumulation of intrapleural air which must be removed as rapidly as it accumulates, minute by minute and second by second. Failure to do so results in lung collapse, possibly tension pneumothorax, more prolonged and voluminous fluid accumulation, perpetuation of the air leak itself, and an increased likelihood of pleural infection, atalectasis, and other undesirable situations contributing to total patient morbidity. An aggressive approach toward the problem is necessary to secure prompt, even, total, and maintained lung expansion. A prerequisite in this regard is a working knowledge of the theory and technical details of the tube-bottle-suction apparatuses commonly used. A consideration of some of these matters is now presented.

FIG. 3. Number 45 right angle French chest tubes

Chest tubes are inserted to do a job of removing pleural contents. More than cursory thought should be given to their size, position, type, number, and withdrawal.

I. *Size.* Large tubes are desirable. They have sufficient rigidity to avoid being kinked, twisted, or otherwise obstructed on changes of the patient's position. They are less easily obstructed by fibrin, clots, or thick fluid. Number 40 or 45 French tubes are recommended (fig. 3). It is questionable whether this size tube is more painful than a smaller one. Usually, however, too much is at stake to entrust the vital drainage function to a small tube.

II. *Position.* A tube can be placed at almost any point in any interspace, even in the first interspace posteriorly. Certain areas are technically awkward or subjectively uncomfortable, such as the region under or very near the scapula, and the posterior interspaces generally, where the tube is painful because the patient lies on it. In general, the preferable area is the lateral chest, between the folds of the latissimus dorsi and the pectoralis muscles. Here the tube passes through a minimum of muscle (or none), and is reasonably comfortable to the patient, permitting him to turn without obstructing it.

III. *Type.* The important consideration, however, is not the point where the

FIG. 4. Roentgenogram taken 10 days after untreated traumatic hemopneumothorax, illustrating apical air loculation and basal fluid collection. This is a common pattern of inadequate postoperative lung expansion and demonstrates the need for two chest tubes.

tube emerges cutaneously, but the point in the pleural space where drainage and suction are being applied. A consideration of gravity and customary patient positions explains the usual pattern of inadequate expansion, which consists of the finding of air in the upper chest, predominantly anteriorly, and fluid in the lower chest, predominantly posteriorly (fig. 4). When both air and fluid are expected to accumulate, it is best to place two tubes in the chest, one with its tip in the very dome of the pleura, the other with its tip in the paravertebral gutter about 2 inches above the diaphragm. A tube placed in the midthoracic region is generally not strategically positioned for the removal of either air or fluid. To accomplish the theoretic objectives of convenient cutaneous position of the tube and strategic placement of the internal end of the tube, right angle catheters are most useful (fig. 3, 5).

IV. *Number.* There should be no hesitancy in putting more than two tubes in the chest. They are easy to insert at the time of surgery, and are simple to remove. Multiple tubes may be indicated in cases where numerous air leaks, uneven expansion, and pleural loculation may reasonably be expected to occur, as for example after an extensive decortication. Simple mushroom catheters are also frequently useful in handling many air or fluid collections, especially those that are recurrent or are less awkwardly located. In general, the inner tip of any tube should be placed at the point at which the expanding lung is last expected to come into contact with the parietal pleura.

V. *Withdrawal.* Chest tubes should be removed when they are no longer

performing the useful function of removing air or fluid, cannot be made to do so, or cannot reasonably be anticipated to do so. In uncomplicated cases, this means from the second to the fifth day postoperatively, allowing 24 hours after the last significant drainage of air or fluid is noted. Patients with persistent air leak must remain intubated until the leak ceases, or until impending infection forces an alteration of total management. A tube that is irretrievably obstructed by a clot may need to be removed and replaced. If the inner tip of a tube lies adjacent to a pleural space but is obstructed by expanded lung, it should be removed and a new tube appropriately inserted where needed. Many other workaday problems of this nature are encountered but can usually be handled by application of the general rule mentioned above.

Several types of tubing-bottle system are in use. The theoretically soundest type is the three bottle system described by Howe,[3] because it takes into account the sudden increase in pleural pressure and volume flow of air from the pleura occasioned by a cough. This advantage is gained, however, at the cost of increased complexity of the system which renders it slightly impractical for routine use.

A widely used system is that depicted in figure 6. The suction created by the pump is transmitted to the air layer in pressure regulating bottle A, thence to the air layer in drainage bottle B (or C), thence via the bottle B (or C) liquid layer and the tube into the chest. Certain points to be kept in mind about this system are as follows:

1. The negative pressure in the system cannot exceed the underwater length of the safety tube in Bottle A.

2. The safety tube in bottle A should bubble constantly. Lack of bubbling means (see below) that the volume per minute air leak from the lung exceeds the volume per minute pumping capacity of the pump, and no effective negative pressure is present in the system.

3. Ejection of water out the top of the safety tube in bottle A, as may occur during coughing, is an ominous sign and signifies a large pneumothorax or a large lung leak or both, plus again the fact that one has an inadequate pump which is

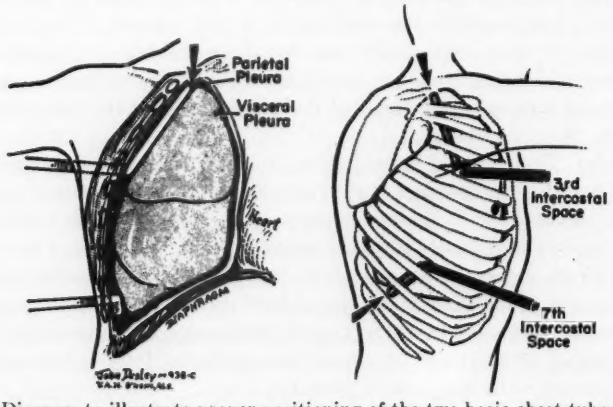


FIG. 5. Diagram to illustrate proper positioning of the two basic chest tubes, the upper one to remove air, the lower one to remove fluid.

incapable of handling momentarily the volume of air which is ejected from the pleura out into the system during the cough. In this circumstance the pump is actually an obstruction in the circuit. The situation is a common cause of improper lung expansion, and has been known to cause tension pneumothorax.

4. Fluctuation of liquid in the glass tube in bottle B (or C) is in direct proportion to the size of the pleural space entered by the tip of the chest tube.

5. As liquid drains into bottle B more of the glass tube is submerged thereunder. The underwater length of the glass tube in bottle B must be subtracted

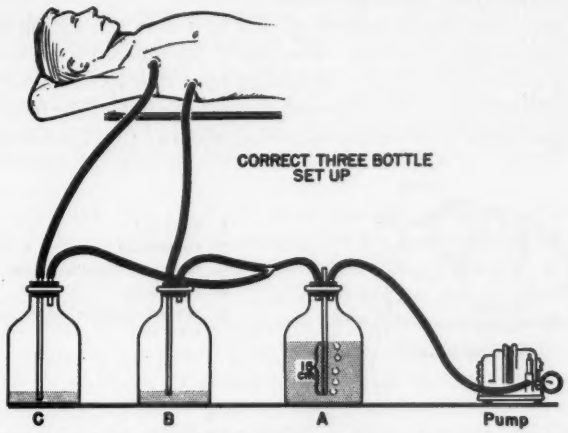


FIG. 6. Diagram of the basic two-tube drainage-suction set-up. See text.

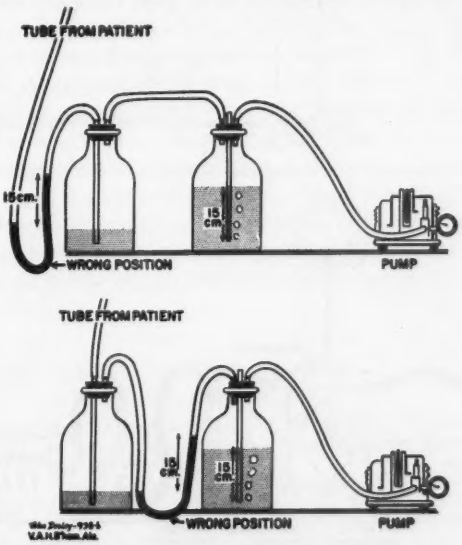


FIG. 7. Diagram to illustrate how a fluid-containing dependent loop in the tubing can destroy suction and hamper drainage.

from the underwater length of the glass tube in bottle A to determine the amount of effective negative pressure which is extant in the pleural space. Thus if pressure regulating bottle A is set for 15 cm. negative pressure, but 13 cm. of the glass tube in bottle B is underwater, the net effective suction is −2 cm. When two chest tube-drainage bottles are used, the net effective negative pressure in each chest tube is the difference between the underwater length of the glass tube in bottle A and the underwater length of the glass tube in whichever drainage bottle has accumulated the most drainage. These problems of pressure loss are solved by inserting fresh drainage bottles.

6. There are many simple sources of obstruction in the tubing systems. Attention to these details, however, often spells the difference between a benign postoperative course and a poorly expanding lung with its attendant morbidity. One important and frequently neglected detail is that the tube from the patient to the drainage bottle should run downhill all the way (fig. 7). A dependent loop

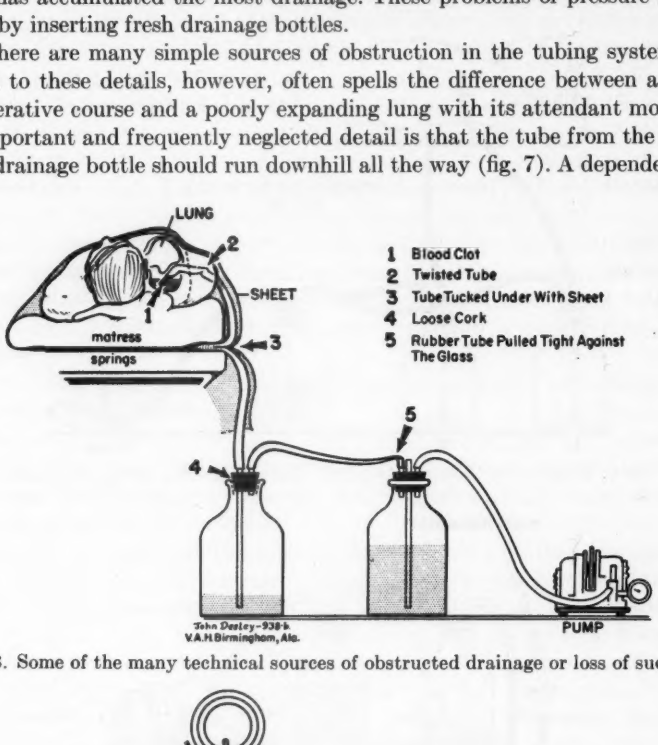


FIG. 8. Some of the many technical sources of obstructed drainage or loss of suction.

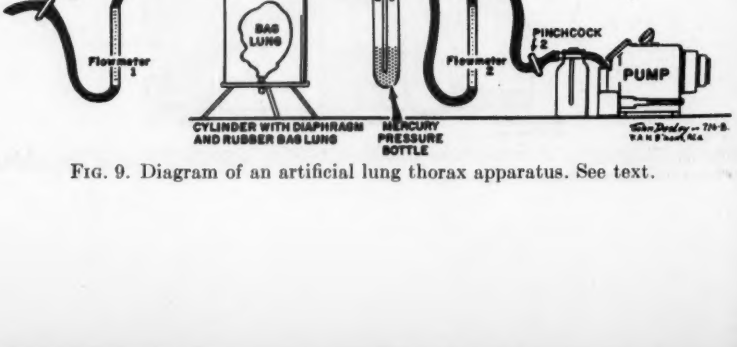


FIG. 9. Diagram of an artificial lung thorax apparatus. See text.

in this tube accumulates fluid which destroys the suction, and even renders it necessary for the patient to build up a positive intrapleural pressure to force the dependent fluid around the loop and uphill into the drainage bottle. Others of the many sources of obstruction or loss of suction are shown in figure 8.

SUCTION

The question of suction should be considered from the standpoint of its two components, pressure and volume flow. There are differing opinions concerning the actual degree of negative pressure that should be delivered to the pleural space, with advocates of pressures ranging up to -40 cm. H_2O. A reasonable answer to this problem would seem to be that one should use a pressure mildly in excess of the amount required to reinflate a normal atelectatic lung. Since the latter figure should not be more than 10 to 15 cm. H_2O,[4] the customarily used suction is -15 cm. H_2O. In the presence of restrictive disease, such as parenchymal fibrotic changes or a pleural peel however, higher negative pressure may be needed.

The matter of volume flow of air per minute has been given insufficient attention in the past, and inadequate lung expansion has been treated by increasing the negative pressure in the system, rather than by using a pump capable of removing an adequate volume flow. The problem is relevant only in cases of intrathoracic air leak. In order to secure expansion, it is necessary that the pump be capable of removing a volume of air per minute exceeding the volume per minute air leak. In addition, the pump should be capable, as mentioned above, of handling the instantaneous increase of ejected air which may occur during a cough. To test some of these theories, the apparatus depicted in figure 9 was constructed. It is an artificial lung-chest set-up, the lung having an inflation resistance of 9 cm. H_2O, and there being an adjustable volume per minute intrathoracic air leak and an adjustable volume per minute pump, both being measured by flowmeters. It was found that if the volume per minute air leak was less than the volume per minute capacity of the pump, that the lung expanded readily at a pressure of -10 cm. H_2O in the system, and that increase in the pressure did not expedite this. It was then found that if the volume per minute air leak exceeded the volume per minute capacity of the pump, that the lung could not be inflated with any degree of negative pressure in the system. Thus the important feature of the pump should be the ability to deliver high volume at low pressure. This fact is currently being recognized by manufacturers of thoracic suction pumps.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Creech, O., De Bakey, M. E., and Amspacher, W. H.: Effect of streptokinase and streptodornase on wound healing in pulmonary resection; experimental study, S. Forum (1951) *2:* 25, 1952.
2. Goehring, W. O., and Grant, J. J.: Allergic reaction to streptokinase-streptodornase solution given intrapleurally, J. A. M. A. *152:* 1429 (Aug. 8) 1953.
3. Howe, B. E., Jr.: Evaluation of chest suction with artificial thorax, S. Forum (1951) *2:* 1, 1952.
4. Lindskog, G. E., and Bradshaw, H. H.: Reinflation of atelectatic lung : experimental study, J. Thoracic Surg. *3:* 333 (April) 1934.

ABERRANT PANCREATIC TISSUE IN GASTRIC WALL: REPORT OF FOUR CASES SIMULATING PEPTIC ULCER

JOE W. DENSON, M.D.

Birmingham

Since Klob[9] first called attention to aberrant pancreatic tissue in 1859, numerous case reports have appeared in the literature with excellent reviews of the subject by Branch and Gross,[2] Faust and Mudgett,[6] Barbosa, Dockerty and Waugh,[1] Busard and Walters,[3] Pearson,[12] Marshall and Curtiss[11] and Wagner.[13]

The largest series from a single source was reported by Barbosa and associates[1] at the Mayo Clinic. Of their 41 authenticated surgical cases, 25 were clinically significant. As stressed by these authors, aberrant pancreatic tissue can no longer be considered an anatomic curio or medical rarity, being found by them once in every 500 operations in the upper abdomen. Every surgeon operating on the gastrointestinal tract should be familiar with its existence, its protean symptomatology, its variance of location, and its pathologic potentialities in order to properly handle the condition when encountered.

Pancreatic tissue occurring outside its usual location without vascular or neural supply from the main pancreatic gland is thought to represent an embryologic variation of growth, rotation of fusion of the three primary anlagen, the dorsal and right and left ventral.

Horgan[7] proposed that aberrant pancreatic tissue resulted from displacement of pancreatic cells by embryonal adhesions in which these cells attach themselves to mesentery, stomach or intestines as they grow in opposite directions. Warthin[14] postulated there was a snaring off of pancreatic tissue as the lateral rudimentary pancreatic ducts penetrate the intestinal wall with migration of this tissue up or down the intestinal tube as it grows longitudinally. Lordy's[10] explanation of this accessory tissue was incomplete regression of the left ventral anlage which normally atrophies.

Aberrant pancreatic tissue is usually found in 1 or 2 per cent of autopsies with reports varying from 0.55 to 5.6 per cent. The incidence seems to be proportional to the diligence with which one searches for the lesion. We have encountered two lesions at re-exploration which had been previously overlooked, one in the duodenum and one in the jejunum. The most common location for this ectopic tissue is in the submucosa of the gastrointestinal tract but it is also found in the muscularis and less frequently beneath the serosa.

Aberrant pancreatic tissue has been reported in the wall, as well as diverticula of the stomach, duodenum, jejunum, and ileum, in Meckel's diverticulum, spleen and its capsule, gallbladder, cystic and common duct, liver, peripancreatic tissue and mediastinal teratomatous cyst. Approximately 70 per cent of the nodules will be in the duodenum, stomach and jejunum in that order of frequency.

The size may vary from that of a microscopic nodule to a tumefaction 4 to 5

From the Veterans Administration Hospital, Birmingham 3, Alabama.

centimeters in diameter. The tissue is firm, yellowish grey, and when located beneath the serosa, it is well demarcated and has a lobulated appearance with a granular surface. The larger nodules have ducts that will admit a probe in a central pit which grossly is not unlike the ampulla of Vater. Serial sections of the smaller nodules often will show an excretory duct opening into the lumen of the adjacent viscera.

Histologically, acinar tissue, ductal epithelium and islet of Langerhan may be identified. Any one of these types of tissue may be predominant. There is usually considerable smooth muscle surrounding the nests of pancreatic tissue. Islet tissue is present in about one-third of the nodules.

Zymogen granules are present in the acinar cells and this tissue is thought to be physiologically capable of secreting pancreatic enzymes. Documented case reports attest the ability of the islet cells to function, producing hypoglycemia and "*Wipple's essential triad*" with complete cures resulting after removal of the aberrant islet cell tissue.

Aberrant pancreatic tissue may undergo any of the pathologic changes common to the normally located pancreas. Acute and chronic inflammation have been described giving rise to acute pancreatic edema, acute hemorrhagic pancreatitis with fat necrosis, abscess and cyst formation. Cystic fibrosis in aberrant tissue and in the normally situated pancreas has occurred concomitantly. It may undergo neoplastic changes. There are case reports of cystoadenocarcinoma, metastasizing insular carcinoma, pancreatic duct carcinoma, malignant adenoma and adenocarcinoma arising in aberrant tissue. This tissue should be considered as a premalignant lesion and as stressed by Barbosa and associates,[4] it is probably the origin of many undifferentiated carcinomas of the duodenum.

The symptoms produced by aberrant pancreatic tissue are as variable as its location. It may produce symptoms by mechanical interference with the organ involved and has been incriminated as the leading point in intussusception in the jejunum and ileum. Pyloric obstruction can be produced by this tissue due to inflammation, scarring and edema in the region of the pylorus or "*ball valve*" type of obstruction in which the polypoid mass in the pre-pyloric region prolapses through the pyloric orifice. Duodenal obstruction has been attributed solely to this tissue. Common and pancreatic duct obstruction may result when the nodule is strategically located in the papilla of Vater. Symptoms indistinguishable from peptic ulcer (both acute and chronic), gallbladder disease, and appendicitis are described which could be attributed only to the aberrant pancreatic tissue. Massive gastric hemorrhage, necessitating emergency laparotomy, has occurred. Hudock and associates[3] reported massive hemorrhage in 5 of 130 collected cases in which the aberrant tissue was found in the stomach of the patient at operation.

Aside from symptoms produced by pure mechanical interference, symptoms are also produced by liberation of enzymes and hormones from the functioning aberrant tissue. Acute hemorrhagic pancreatitis occurring in a nodule in the gastrohepatic ligament and producing fat necrosis support the fact that tryptic activity is present in the aberrant tissue. Cave[4] in 1928, noted that ingestion of meat aggravated the symptoms produced by this tissue which was thought to

be due to the fact that proteins act as a strong stimulus to pancreatic secretion. Functioning extrapancreatic islet cell adenomas have produced hypoglycemia even to the point of coma with complete relief of symptoms after surgical removal of the functioning aberrant tissue.

Waugh and Harding[15] presented an excellent discussion of the clinical significance of heterotopic pancreatic tissue in the pyloric orifice and they outlined five different ways in which it could produce serious consequences:

1. Interference with passage of food through the stomach by prolapse of the polypoid excrescence through the pyloric orifice.

2. Although there may be no actual stenosis, the presence of this tissue in pyloric orifice can interfere with its peristaltic activity.

3. Peptic ulcer actually arises from the discharge of pancreatic juice through the duct in its base. Four cases were presented in which ducts from the aberrant tissue were seen microscopically to enter the base of the ulcer.

4. Even though an ulcer is not present, the possibility that the activity of the tissue or its excretion may cause functional disturbances.

5. Malignant and benign neoplasm may arise from such aberrant tissue.

In the past 3 years we have encountered 4 cases that presented the classical symptoms of peptic ulcer in which only aberrant pancreatic tissue was found in the gastric wall to account for the symptoms.

CASE REPORTS

Case no. 1. J. L. C., a 24 year old white man, was admitted to the Birmingham Veterans Administration Hospital on Feb. 4, 1954, with a history of *"stomach trouble"* for the past 6 years. He began having crampy upper abdominal pain associated with nausea and vomiting while abroad ship in 1948. He was hospitalized for 3 months with a diagnosis of *"duodenal ulcer"*. He improved on ulcer therapy but never became asymptomatic. For the next 6 years he was hospitalized 3 times for similar symptoms together with episodes of vomiting lasting from a few hours to a day or so in which he was unable to retain anything on his stomach. For 2 weeks prior to admission he had vomited almost everything ingested including his medications.

General physical examination was negative on admission except a moderate amount of tenderness in the midepigastrium. Laboratory Data: Admission white blood cell were 8,450 per cu. mm. with normal differential; hemoglobin 16 grams; urinalysis negative; total protein 7.4 with normal A/G ratio; prothrombin time 100 per cent; blood urea nitrogen 22.5 mg. per cent; stool specimen positive for blood. The overnight 12 hour gastric secretion was 1,650 cc. with 14.3 mEq. of free and 20.2 total acid. A repeat showed a total volume of 1,550 cc. with 32 mEq. free acid and 41 total acid. Cholecystogram showed a normal functioning gallbladder. Gastro-intestinal series showed a constant irregular deformity of the duodenal cap without spasm or irritability.

On Feb. 24, 1954, abdominal exploration was carried out. A tumefaction was palpable in the prepyloric area on the greater curvature side just above the pyloric orifice. The duodenum appeared normal and was freely movable, with no evidence of scarring. A gastrotomy was done and a polypoid mass about 3 cm. in diameter was noted with two openings into the lumen of the stomach. The mass could be made to prolapse easily through the pyloric orifice. The impression at this time was aberrant pancreas versus leiomyoma. Because of high acid values a subtotal gastrectomy with gastroenterostomy was carried out.

The pathologic report was as follows: At a point 8 mm. above the pylorus there is a large polypoid mass with two openings on the surface, grossly resembling the ampulla of Vater, which admit a probe 1.5 cm. and 1 cm. respectively. The firm nodule measures 2.8 by 1.2 cm. and is made up of dense pearly grey tissue. Adjacent to this tissue is a cyst measuring 0.5 cm.

in diameter and filled with a clear amber fluid. Microscopic sections showed pancreatic tissue, both acinar and islet, with a preponderance of acinar tissue. Adjacent to this tissue is a small cyst with no epithelial lining.

This patient's symptoms were thought to be due to prolapse of the aberrant pancreatic tissue through the pylorus giving rise to intermittent pyloric obstruction. Except for mild symptoms of "*dumping*" for 2 months after subtotal gastrectomy he has remained asymptomatic.

FIG. 1 A. Case 1 A. Section of polypoid nodule showing 2 central pits into which pancreatic ducts empty. Magnification × 30.

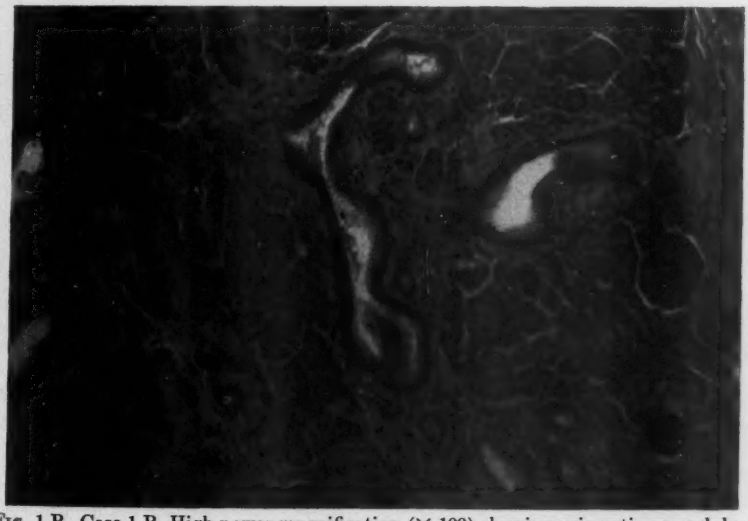


FIG. 1 B. Case 1 B. High power magnification (× 100) showing acinar tissue and ductal epithelium.

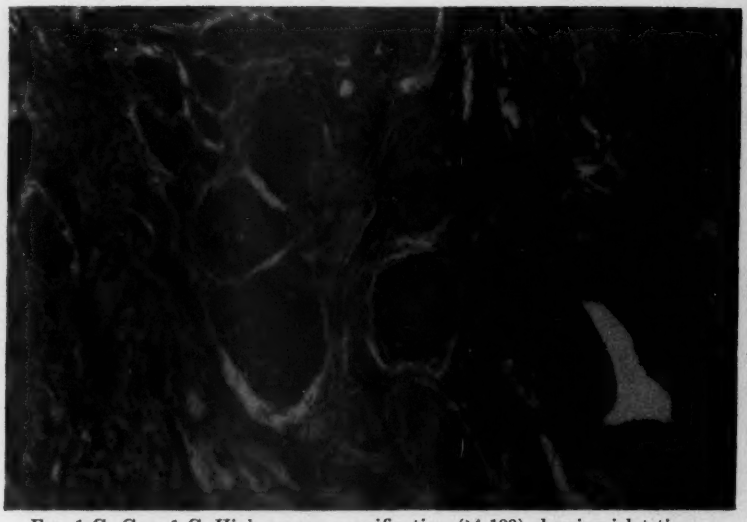


FIG. 1 C. Case 1 C. High power magnification (× 100) showing islet tissue

Case no. 2. J. H. M., a 54 year old white man, was first admitted to the Medical Service of the Birmingham Veterans Administration Hospital on Feb. 26, 1954, because of pain in the upper abdomen for a duration of 7 months. The pain was described as burning epigastric pain relieved by food and amphojel. There was one episode of hematemesis prior to admission.

A gastrointestinal series revealed a small ulcer crater in the prepyloric area on the lesser curvature side. The patient was gastroscoped and the ulcer visualized and thought to be benign. He was placed on ulcer therapy with disappearance of the ulcer crater radiographically and evidence of healing when regastroscoped.

The patient was readmitted on May 24, 1954, for follow-up studies with no evidence of ulcer by gastrointestinal roentgenograms or gastroscopic examination.

He was admitted for the third time on Jan. 3, 1955, because of a recurrence of epigastric pain for approximately 1 month prior to admission. A gastrointestinal series showed a prepyloric ulcer.

An overnight 12 hour gastric secretion revealed a volume of 1,100 cc. with no free hydrochloric acid and 77.5 mEq. of total acid. On Jan. 24, 1955, a laparotomy was performed. The duodenum appeared normal to inspection and palpation. There was slight induration and scarring in the prepyloric area. A subtotal gastrectomy with gastrojejunostomy was carried out. On gross description of the surgical specimen there were two small superficial ulcerated lesions, one 4 cm. from the pylorus, and another 2 cm. from the pylorus near the lesser curvature side. Microscopic examination revealed chronic inflammatory cell reaction in the mucosa and submucosa at the pyloric area with pancreatic duct-like structures in the submucosa of this area. No acinar tissue was identifiable.

The patient had remained asymptomatic since the time of the operation.

Case no. 3. E. L. H., a 34 year old white man, entered the Birmingham Veterans Administration Hospital Dec. 22, 1955, with a history of episodes of burning epigastric pain since World War II. The pain was relieved somewhat by ingestion of food and alkali.

Approximately 2 months prior to admission he vomited practically everything he ate. Twelve days before admission he vomited considerable amounts of blood and tarry stools were noted 2 days later. He was admitted to a local hospital and given 5 transfusions of

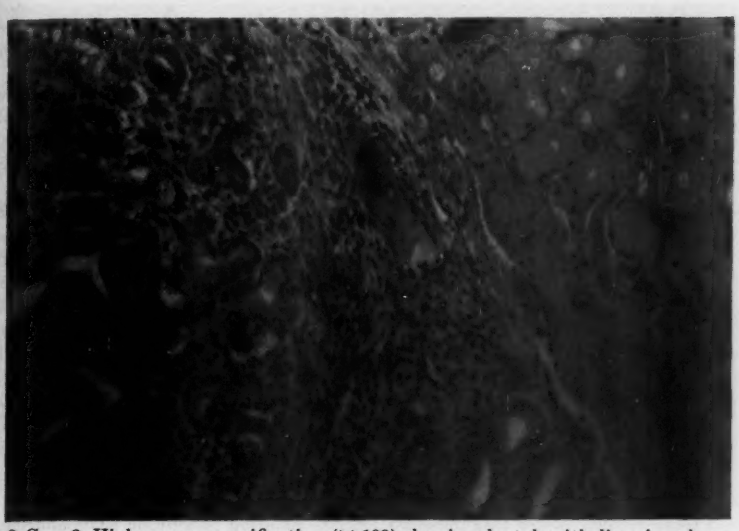


FIG. 2 Case 2. High power magnification (× 100) showing ductal epithelium in submucosa

whole blood. A gastrointestinal series was obtained which showed almost complete pyloric obstruction. The patient was then transferred to the Birmingham Veterans Administration Hospital after 1 week of hospitalization.

Laboratory Data: white blood cells were 5,200 per cu. mm with normal differential; hemoglobin 68 per cent; hematocrit 38 mm.; total protein 6.6 with normal A/G ratio; prothrombin time 100 per cent; 3 per cent bromsulphalein retention; serum amylase 68 mg per cent; blood urea nitrogen 13 mg. per cent; urinalysis negative; 12 hour overnight gastric secretion showed total volume of 1,200 cc. with 13.4 mEq. free hydrochloroic acid and 93.6 mEq. total acid.

The gastrointestinal series revealed a large and atonic stomach with a prepyloric deformity with minimal patency of the pylorus.

The patient was placed on nasogastric suction for several days along with atrophine and phenobarbital. He was then carried on a strict ulcer regime of milk and cream every hour which he tolerated well.

On Jan. 23, 1956, the patient was explored and there was marked scarring with partial stenosis at the pylorus. A Polya-Hofmeister subtotal gastrectomy was carried out.

Pathologic report of the surgical specimen was reported as showing aberrant pancreatic tissue, ductal and acinar, with chronic inflammatory reaction which extended throughout all layers in the pyloric area. The pancreatic tissue was located in the submucosa. A small superficial ulceration was found in the prepyloric area.

The patient made an uneventful recovery and has been asymptomatic since.

Case no. 4. S. E. M., a 65 year old white man, entered the Birmingham Veterans Administration Hospital on Jan. 9, 1956, with a history of epigastric distress for a 12 year duration, described as a burning, gnawing pain coming on 2 to 3 hours postprandially, and relieved by milk and *"soda and aspirins"*. In 1951, he sought medical aid, was told he had a *"duodenal ulcer"* and was treated with diet, phenobarbital and banthine with partial relief of symptoms. Three months prior to admission his pain became more severe and radiated *"straight through to his back"*.

On admission, general physical examination was negative except for a hypertension of 198/110.

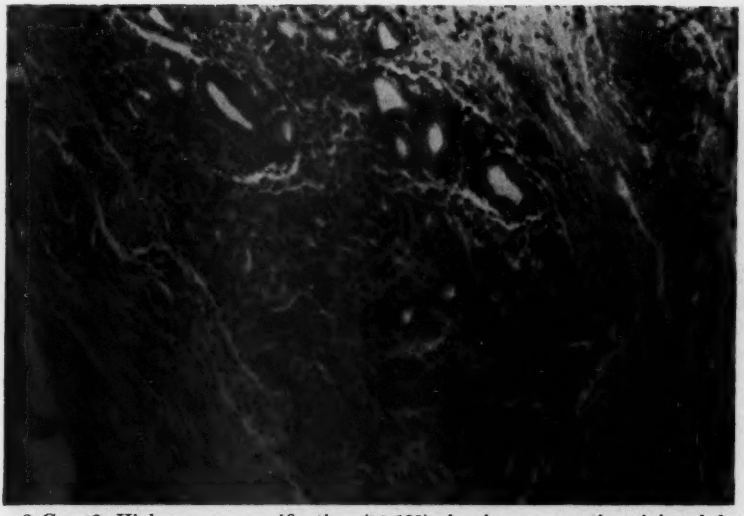


FIG. 3 Case 3. High power magnification (× 100) showing pancreatic acini and ductal epithelium.

FIG. 4 Case 4. High power magnification (× 100) showing pancreatic acini and ductal epithelium.

Laboratory Data: Admission urinalysis was negative; white blood cells were 9,700 per cu. mm. with normal differential; hemoglobin 14 grams; hematocrit 45 mm.; blood urea nitrogen 34 mg. per cent; serum amylase 34 mg. per cent; stools negative for occult blood; 12 hour overnight gastric secretion revealed a total volume of 1,200 cc. with 72.6 mEq free hydrochloric acid and 101.6 mEq. total acid.

Gastrointestinal series showed a prepyloric defect with edematous posterior bulbar

mucosa and deformity of the duodenal bulb. There was a small ulcer on the anterior superior surface.

The patient was placed on a strict ulcer regime with some improvement but continued to have pain. He was discharged on medical therapy but readmitted to surgical service on March 1, 1956, because of intractable pain. Another gastrointestinal series showed a small ulcer in the fundus of the stomach and considerable edema in the prepyloric area. The duodenal bulb was deformed but no active ulcer was identified.

On March 12, 1956, exploration was carried out. The duodenum was found to be free and pliable with no evidence of scarring. The pylorus was markedly edematous with partial stenosis and a small nodular tumefaction was palpable in the pylorus. A subtotal gastrectomy was carried out.

Gross description of the surgical specimen was reported as showing flattening of mucosal folds in prepyloric area and scarring in pyloric region. The circumference of the pylorus measured only 5 mm. Microscopic sections showed chronic inflammatory cells throughout all layers of the pyloric area with aberrant pancreatic tissue composed of ductal epithelium and pancreatic acini, extending from pylorus into duodenal cuff. The ducts from the aberrant pancreatic tissue could be seen opening directly into the lumen of the duodenum.

The patient made an uneventful recovery and has remained asymptomatic since the operative procedure.

DISCUSSION

It is well known that there are many instances in which aberrant pancreatic tissue had produced no symptoms whatsoever and if the theory of congenital origin is adhered to, it is difficult to explain the fact that symptoms most frequently become manifest in the fourth, fifth, and sixth decades of life. However, if one keeps in mind the pathophysiology of this aberrant tissue, it can be seen how symptoms could be readily attributed solely to its presence.

It is not known whether the pancreatic enzymes cause ulceration by direct digestive action on the mucosa or whether the tissue itself is less resistant to acid peptic digestion than the normal surrounding tissue. It seems feasible that the alkaline pancreatic secretions from this aberrant tissue flowing directly into the gastric antrum could cause liberation of gastrin by the antrum which in turn cause secretion of hydrochloric acid in the fasting stomach and lead to acid peptic digestion.

All of our patients had high volumes of overnight 12 hour gastric secretion and 3 of the 4 had hyperchlorhydria. According to Waugh and Harding,[15] hypochlorhydria is most often associated with this aberrant tissue but only 1 case of achlorhydria was recorded.

Our interest in aberrant pancreatic tissue in the gastric wall was stimulated by Zollinger and Ellison's[16] report of peptic ulceration associated with noninsulin producing islet cell adenomas of the pancreas. They postulated a hormonal or chemical factor arising in the pancreatic adenoma that is capable of stimulating the gastric glands to secrete and in turn to be the causative agent of peptic ulcer disease. This view is supported by documented observation of the return of the gastric secretions to normal levels following resection of a noninsulin producing islet cell tumor of the pancreas in a patient suffering from hypersecretion and recurrent marginal ulceration.

It seemed possible that aberrant pancreatic tissue in a like manner could be

capable of producing peptic ulceration by functioning nonbeta cells, although to a less degree than that associated with the nonbeta cell adenomas located in the normally situated pancreas.

Ellison[5] recently reported on 24 collected cases of ulcerogenic tumors of the pancreas. Of these, 1 was a cystic islet cell carcinoma reported by May in 1937, arising in aberrant pancreatic tissue and producing gastric ulceration.

SUMMARY

A brief review of the literature of aberrant pancreatic tissue is reported.

Four cases presenting classical ulcer symptoms are described in which only aberrant pancreatic tissue was found to explain the symptoms.

The mechanism of production of symptoms from this aberrant tissue is discussed.

V. A. Hospital
Birmingham 3, Ala.

REFERENCES

1. Barbosa, J. deC., Dockerty, M. B., and Waugh, J. M.: Pancreatic heterotopia; review of literature and report of 41 authenticated surgical cases of which 25 were clinically significant, Proc. Staff Meet., Mayo Clin. *21:* 246 (June 26) 1946.
2. Branch, C. D., and Gross, R. E.: Aberrant pancreatic tissue in gastrointestinal tract, Arch. Surg. *31:* 200 (Aug.) 1935.
3. Busard, J. M., and Walters, W.: Heterotopic pancreatic tissue, Arch. Surg. *60:* 674 (April) 1950.
4. Cave, H. W.: Aberrant pancreatic tissue simulating duodenal ulcer, Am. J. Surg. *5:* 407 (Oct.) 1928.
5. Ellison, E. H.: Ulcerogenic tumor of pancreas, Surgery *40:* 147 (July) 1956.
6. Faust, D. B., and Mudgett, C. S.: Aberrant pancreas with review of literature and report of case, Ann. Int. Med. *14:* 717 (Oct.) 1940.
7. Horgan, E. J.: Accessory pancreatic tissue; report of 2 cases, Arch. Surg. *2:* 521 (May) 1921.
8. Hudock, J. J., Wanner, H., and Reiley, C. J.: Acute massive gastrointestinal hemorrhage associated with pancreatic heterotopic tissue of stomach, Ann. Surg. *143:* 121 (Jan.) 1956.
9. Klob, J.: cited by Branch, and Gross.[2]
10. Lordy, C.: cited by Barbosa, Dockerty and Waugh.[1]
11. Marshall, S. F., and Curtiss, F. M.: Aberrant pancreas in stomach wall, S. Clin. North America *32:* 867 (June) 1952.
12. Pearson, S.: Aberrant pancreas, review of literature and report of 3 cases, 1 of which produced common and pancreatic duct obstruction, Arch. Surg. *63:* 168 (Aug.) 1951.
13. Wagner, M.: Clinical significance of heterotopic pancreas in wall of stomach, report of case and review of literature, Arch. Surg. *64:* 840 (June) 1952.
14. Warthin, A. S.: Two cases of accessory pancreas, Phys. Surg. *26:* 337, 1904.
15. Waugh, T. R., and Harding, E. W.: Heterotopic pancreatic tissue in region of pyloric orifice, Gastroenterol. *6:* 416 (May) 1946.
16. Zollinger, R. M., and Ellison, E. H.: Primary peptic ulceration of jejunum associated with islet cell tumors of pancreas, Ann. Surg. *142:* 709 (Oct.) 1955.

FAMILIAL POLYPOSIS OF THE COLON: THE FATE OF THE RETAINED RECTUM AFTER COLECTOMY IN CHILDREN

T. Brannon Hubbard, Jr., M.D.

Montgomery

Familial polyposis of the colon is that entity in which there is an inherited instability of the epithelial cells of the large bowel resulting in a pathologic proliferation of the epithelial lining, producing intraluminal folds of varying shapes termed polyps. These may exist as villous, sessile, or pedunculated structures, and usually stud the colon almost confluently from ileocecal valve to anus.[21, 15] The disease is transmitted as a Mendelian dominant without regard to sex, with occasional lack of penetrance of the dominant gene.[24] As a general rule the polyps become clinically apparent about the age of 20 years, malignant changes develop about 15 years later, and untreated patients die from cancer in their early forties.

In the last 15 years reports of such cases have become commonplace and the treatment has been standardized to the extent that everyone agrees that, when such a case is diagnosed, a colectomy should be performed due to the inevitable development of malignant degeneration. Whether or not to preserve the rectum is as yet not decided unanimously. There are those who believe the milder cases should have the rectum preserved, the rectal polyps fulgurated, and careful follow-up carried out.[12, 32, 23] There are others who believe that the development of cancer in the rectal stump is so inevitable that the rectum should be excised and ileostomy performed.[3] Certainly there is abundant evidence in the literature that further polyps and ultimately cancer may appear in the retained rectum. However, there is no reported series which presents a group of patients followed to 40 years of age or more, so no definite percentages can be quoted. It may be said that new polyps have occurred in the rectal stump in almost all patients, and that cancer has occurred in 4 per cent to 16 per cent of the patients.[13, 12, 32]

The above discussion refers to patients grouped together regardless of age, and it has been generally assumed that the polyps do not often appear until the age of 20 years or thereabouts. However, a glance at the literature shows that cases in children are not a great rarity. For example, Dukes reported three families in which he studied the children carefully, and of a total of 22 children, ranging in age from 2 to 15 years, there were 7 who presented polyposis.[10] Le Fevre has reported an instance of extensive polyposis in a 3 month old child.[20]

The purpose of this paper is to describe the case of a 9 year old boy whose disease has presented some unusual developments.

CASE REPORT

This 9 year old white boy was first seen on Nov. 12, 1953. His mother, upon whom we had previously performed a colectomy, presented him with the story that all his life his bowels

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

had moved from 4 to 10 times a day. Moreover, as soon as he ate, he had always had an immediate need to defecate. For the past 4 or 5 months, occasional bright blood had been noticed in the stools. Except for the embarrassment of having to leave his classroom frequently, development had been essentially normal.

His family history revealed that his maternal great-grandfather had died of cancer of the colon at the age of 70 years. His maternal grandmother had died of polyposis and cancer at the age of 37 years. Four maternal uncles had developed polyposis, 3 with malignant degeneration; and the patient's mother had undergone colectomy for diffuse polyposis. In the fourth generation, in addition to the patient, there was a first cousin who had undergone colectomy for polyposis at the age of 18 years. All patients in the third and fourth generations who were not treated by us have been confirmed by the attending surgeon. Those in the first and second generations have been attested to only by the members of the family (fig. 1).

Physical examination revealed a fairly well-nourished white boy, weighing 67.5 pounds and of above average mental development. Positive findings were limited to the proctoscopic examination which revealed a rectal mucosa completely studded with small polyps, extending from the pectinate line upward. No blood, ulceration, or inflammation was apparent. The polyps measured from 1.0 to 6.0 millimeters in thickness, from 2.0 to 5.0 millimeters in height, and were predominantly sessile in type. Little or no normal mucosa was visible.

Barium enema with air contrast studies revealed extensive polyposis, predominantly in the distal half of the colon but also in the right colon (fig. 2). Other pertinent laboratory findings were a hemoglobin of 13.0 grams, a leukocyte count of 10,700 per cu. mm. with 54 per cent lymphocytes, 43 per cent neutrophiles, and no esoinophiles.

Operation was scheduled for the following spring. Stool examination on March 10, 1954 and again on March 17, 1954 revealed E. Hystolytica trophozoites, E. Coli trophozoites and cysts, E. Nana trophozoites and cysts, and D. Fragilis trophozoites. He was placed on Terramycin 50 mg. per kilogram per day, but this was discontinued after 3 days due to nausea. He was then placed on Milibis-Aralen, 1 tablet three times a day. This was continued for 1 week, discontinued for 1 week, and then given again for 1 more week. At this point the stool was free of parasites.

Operation was performed on April 21, 1954. The colon was removed from the ileocecal valve to within 13 centimeters of the pectinate line as measured by a rectal tube. The rectal stump when opened revealed a mucosa studded with almost confluent polyps. A mucosal suture would have been troublesome, but with a single layer of seromuscular sutures it was

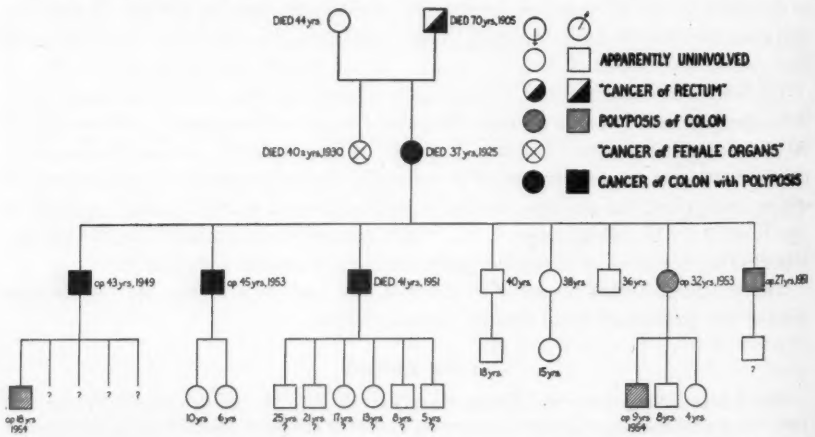


FIG. 1: Pedigree of the family with familial polyposis of the colon reported herein. The patient is designated in the fourth generation.

not difficult to anastomose ileum to rectosigmoid end to end, the ileum having been originally divided flush with the ileocecal valve. The postoperative course was uneventful, the patient being discharged from the hospital on the seventh postoperative day.

Pathologic examination revealed a segment of colon, including cecum and appendix, measuring 63.0 cm. in length after fixation. The serosal surface presented no abnormalities. The mucosal surface was covered with myriad polyps, numbering well over 100 and extending throughout the length of the specimen. These measured from 1.0 millimeter to 5.0 millimeters in diameter. There was no grossly visible ulceration. Microscopic examination revealed numerous small polypoid structures supported by a central core of loose connective tissue and covered by epithelial cells which showed a moderate degree of hyperplasia but no evidence of malignancy. Representative sections are shown in figure 3, contrasted with a relatively uninvolved area of colon and compared with a segment from the colon of the patient's 18 year old first cousin. The similarity between the latter lesions and those of the present case are apparent. Also evident is the lack of any ulceration or scarring, suggesting that these are not pseudopolyps but true neoplasms.

It was planned to fulgurate the remaining rectal polyps by stages. However, due to the patient's age, it was thought that he could be given a few months respite. The first proctoscopic examination was therefore performed on Sept. 18, 1954, 5 months postoperatively. There had occurred, much to our surprise, a considerable decrease in the number of polyps. No fulguration was performed, since we wanted to see what was happening. Proctoscopy was performed again on Jan. 24, 1955, and only 6 polyps were present, the largest measuring 5.0 millimeters in diameter. Again no fulguration or excision was done, and on March 21, 1955, 11 months after colectomy, there were no polyps visible on proctoscopy. As late as Sept. 29, 1956, the rectum appeared completely normal from pectinate line to the ileorectal anastomosis at 13.0 centimeters. The patient has 1 to 3 bowel movements a day, has lost his previous urgency to defecate, and is developing normally.

DISCUSSION

Although one case is of no great significance, it was thought that any unexplained event, which is possibly related to carcinogenesis, should be explored carefully. Two possible explanations for the disappearance of these polyps are apparent.

A. *Were the polyps secondary to the amebic infestation, therefore subsiding when the latter was cured?* It has been thought that the initiating stimulus for the formation of colon polyps is irritation, either infectious or mechanical, leading to inflammation and thence to hypertrophy and hyperplasia.[31] That the amebic infestation in this patient acted as such a stimulus is possible. However, scrutiny of the literature offers neither confirmation nor contraction of this theory. A number of the representative articles and the two leading monographs on amebiasis include a total of 800 personal cases described at autopsy or proctoscopy by the authors, and no mention is made of polyp formation.[2, 7, 9, 11, 26, 27] However, Radke wrote that it had long seemed to him that the incidence of rectal polyp was greatly increased in amebiasis cases over those with no disease, and that he had found "polypoid or pseudopolypoid growths" in the rectosigmoidal area of 7 per cent of 245 cases of amebiasis, as compared with 1 per cent of 200 persons examined by sigmoidoscopy in whom amebiasis did not exist.[26] As a statistical comparison these figures are not too significant since the age range of the two groups is not described. Moreover, the incidence of 1 per cent in the control group is so much below that generally reported for the popula-

FIG. 2A

FIG. 2: Two views of the barium enema roentgenograms of the patient whose case is reported herein, showing extensive polyposis. A few polyps are visible in the rectal ampulla.

tion at large, that its significance is questionable.[18] Third, the inclusion of the term "pseudopolyps" is not germane to the present discussion, pseudopolyps being not true neoplasms but islands of normal mucosa distorted by surrounding ulceration and scarring. Similarly a case reported by Hines in 1923 of extensive polypoid formations in an amebic colon is on careful reading a case of pseudopolyposis.[16]

Two cases of familial polyposis of the colon have been reported in the literature in which the patients had amebae recovered from the stool.[1, 35] These two isolated cases are alone of no great significance, but on the other hand only a few of the patients in the reported cases of polyposis apparently had stool examinations for parasites.

B. *Was the removal of the proximal colon the cause for the disappearance of the polyps?* The idea that polyps (and ultimately cancer) occur rarely in the small

FIG. 2B

bowel but often in the colon because of the irritation of the relatively inspissated and static feces is, of course, not new. As a matter of fact in 1937 Hoelzel and Da Costa reported the development of polyps of the colon in 34 per cent of 250 rats which were fed diets high in inert materials such as kaolin or talcum powder for from 2 days to 6 months.[17] Although one attempt to confirm this work was unsuccessful, the later work may be considered inconclusive since the rats were only kept on the diet for 29 days.[22] In regard to the present case, it is of course tempting to wonder if early colectomy with the abolition of more inspissated, irritating feces was followed in the child by disappearance of the polyps, just as early leukoplakia may sometimes disappear with improvement of mouth hygiene. In this regard the recent statement of Mr. Ian Todd of St. Mark's Hospital is of interest. He said, "I believe it is possible that there is some agent completely unknown that tends to make the polyps grow in the rectum and in the colon, and that having removed the greater part of this organ in some people, they will lose the tendency to form polyps. I know this is getting on very dangerous

FIG. 3A

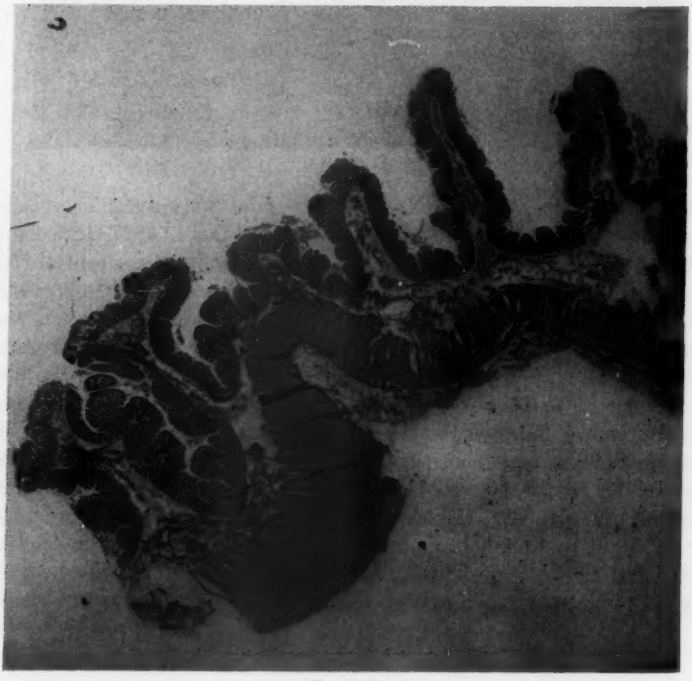


FIG. 3B

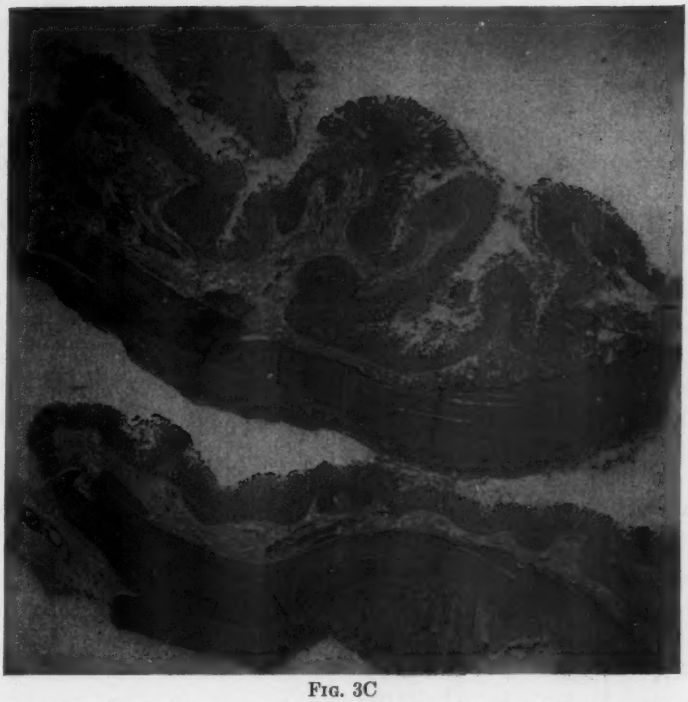


FIG. 3C

FIG. 3: Photomicrographs (lower power) of the excised colon. Figs. 3A and 3B show 2 segments of involved colon compared with a relatively uninvolved segment. Fig. 3C is of the colon of the patient's 18 year old first cousin. The similarity is apparent.*

* We are indebted to the U. S. Naval Hospital, San Diego, California, for the loan of the latter slide.

grounds, but I believe that we have a certain amount of evidence to substantiate this, that there may be some factor stimulating polyp formation."[36]

This theory can only be corroborated by observing the rectum without fulguration in more children who present familial polyposis. However, it was thought that ancillary evidence might be obtained by learning the long-term results in children who have had colectomy and ileosigmoidostomy. Seventeen such cases have been collected from the literature. In all instances the polyps present in the rectal stump at the time of operation were fulgurated or excised. As recent follow-up as possible has been secured through correspondence with the authors, to whom we are indebted for their interest and help. These cases are listed in table I.

It is apparent that 6 of these 17 patients have developed new polyps in the rectal stump and that one (number 15) developed cancer in the rectal stump. Therefore, from a practical point of view, the retention of the rectum in children would seem to entail a risk similar to that in the adult. Nevertheless, this finding does not necessarily contradict the theory that in the particular patient whose

TABLE I

Collected cases of colectomy and ileosigmoidostomy in children with polyposis of the colon: the fate of the retained rectum

Author	Age, Sex	Family History	Pathology	Length of Follow-up	Age at End of Follow-up	Result
Coleman and Eckert[8]	10 yrs. male	(+)	No polyps in rectum	1 yr.	11 yrs.	No new polyps
Coleman and Eckert[8]	8 yrs. male	(+)	2 polyps in rectum	2½ yrs.	11 yrs.	No new polyps
Gross[14]	6 yrs. female	(+)	Diffuse	3 yrs.	9 yrs.	No new polyps mentioned
Gross[14]	6 yrs. female	(+)	Diffuse	3 yrs.	9 yrs.	3 new polyps
Potts[25]	9 yrs. male	(+)	Diffuse	3 yrs.	12 yrs.	2 polyps in ileum. Several in retum
Gross[14]	9 yrs. male	(+)	Diffuse	5 yrs.	14 yrs.	2 new polyps
Coleman and Eckert[8]	3 yrs. female	(?)	One polyp in rectum	5 yrs.	8 yrs.	No new polyps
Boehme[5]	12 yrs. male	(+)	Diffuse	6 yrs.	18 yrs.	"2 or 3 tiny polyps" at last examination[6]
Black and Hansbro[4]	7 yrs. male	(?)	Only two polyps in rectum	7 yrs.	14 yrs.	No new polyps
Coleman and Eckert[8]	6 yrs. male	(?)	One polyp in rectum	7 yrs.	13 yrs.	No new polyps
Scarborough[30]	3–6 yrs.	(?)	Diffuse	8+ yrs.	11–14 yrs.	No new polyps[29]
Scarborough[30]	3–6 yrs.	(?)	Diffuse	8+ yrs.	11–14 yrs.	No new polyps[29]
Gross[14]	9 yrs. female	(+)	Diffuse	11 yrs.	20 yrs.	No new polyps mentioned
Gross[14]	7 yrs. male	(−)	Diffuse	12 yrs.	19 yrs.	Occasional new polyps
Guptill[15]	11 yrs. female	(+)	Diffuse	12 yrs.	23 yrs.	Cancer of rectum after 12 years[19]
Rankin and Webb[28]	15 yrs. male	(+)	Diffuse	18 yrs.	33 yrs.	3 new polyps 1937–1941. None since[37]
Soper[33]	8 yrs. male	(?)	7 polyps in rectum; 8 polyps in colon	22 yrs.	30 yrs.	Asymptomatic rectum not mentioned[34]

case is reported in this paper the rectal polyps subsided because of the proximal colectomy. It is possible that with prolonged irritation and further solidification of feces, due to so-called colonization of the ileum, new polyps could still develop later, even though this theory were correct. Moreover, it is imaginable that such a procedure could be effective in some colons and not in others, due perhaps to a difference in genetic susceptibility. Such a case as that reported by Rankin and Webb (table I), in which the patient presented diffuse rectal polyposis before colectomy and who has developed only 3 polyps thereafter, none in the past 15 years, must be explicable in some way.

CONCLUSION

The patient with familial polyposis of the colon presents the clinician with a fortuitous, "pure-bred" strain which shows the purest reactions to those various stimuli, which probably through one form of irritation or another, either infectious or mechanical, cause cancer of the colon. This case is reported because no similar phenomenon has been found in the literature; but also in the hope that, from an investigational point of view, anyone treating such a patient will search for amoebae and, if any are found, eradicate the parasites and observe the result, or, if colectomy is done, preserve the rectum without fulgurating the rectal polyps so that our experience can be confirmed or denied.

From a practical point of view, there are some who believe that the rectal segment should not be preserved if the polyposis is too extensive therein.[8] We believe, based upon this one case, that the rectum should be observed for some time after colectomy has been done, before deciding to sacrifice it.

SUMMARY

A case of familial polyposis of the colon in a 9 year old boy is reported, in whom the rectal polyps disappeared spontaneously after colectomy and ileosigmoidostomy.

The results of colectomy with preservation of the rectum in children with polyposis as reported in the literature are summarized.

The possible implications of the disappearance of the polyps in the present case are discussed.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Allen, J. M., and Seamans, C. M.: Duodenal polyps associated with multiple polyposis of colon, Gastroenterol. *28:* 856 (May) 1955.
2. Anderson, H. H., Bostick, W. L., and Johnstone, H. G.: Amebiasis, pathology, diagnosis and chemotherapy, Springfield, Illinois, Charles C Thomas Co., 1953.
3. Bartlett, R. W., and Peck, M. E.: Management of multiple polyposis of colon, Surg., Gynec. & Obst. *90:* 547 (May) 1950.
4. Black, B. M., and Hansbro, G. L.: Treatment of familial polyposis of colon, S. Clin. North America *30:* 1013 (Aug.) 1950.
5. Boehme, E. J.: Surgical treatment of familial polyposis of colon, Ann. Surg. *131:* 519 (April) 1950.
6. ——: Personal communication, 1956.
7. Clark, H. C.: Distribution and complications of amebic lesions found in 186 postmortem examinations, Am. J. Trop. Med. *5:* 157 (March) 1925.

8. Coleman, S. T., and Eckert, C.: Preservation of rectum in familial polyposis of colon and rectum, A.M.A. Arch. Surg. *73:* 635 (Oct.) 1956.
9. Craig, C. F.: Etiology, Diagnosis and Treatment of Amebiasis, Baltimore, Md., Williams and Wilkins Co., 1944.
10. Dukes, C. E.: Familial intestinal polyposis, Ann. Eugenics *17:* 1 (Jan.) 1952.
11. El-Ghaffar, Y. A., and El-Ghaffar, M. A.: Terramycin in amebiasis, Gastroenterol. *29:* 86 (July) 1955.
12. Everson, T. C., and Allen, M. J.: Subtotal colectomy with ileosigmoidostomy and fulguration of polyps in retained colon; evaluation as method of treatment of polyposis (adenomatosis) of colon, A.M.A. Arch. Surg. *69:* 806 (Dec.) 1954.
13. Flotte, C. T., O'Dell, F. C., Jr., and Coller, F. A.: Polyposis of colon, Ann. Surg. *144:* 165 (Aug.) 1956.
14. Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia and London, W. B. Saunders Co., p. 312, 1953.
15. Guptill, P.: Familial polyposis of colon, Surgery *22:* 286 (Aug.) 1947.
16. Hines, L. E.: Form of polypoid colitis, a late stage of amebic dysentery, J. A. M. A. *81:* 12 (July 7) 1923.
17. Hoelzel, F., and Da Costa, E.: Experimental production of polyposis of colon in rats, Am. J. Dig. Dis. & Nutrition *4:* 23 (March) 1937.
18. Hubbard, T. B., Jr., and State, D.: Cancer detection, report from the Center at the University of Minnesota, Minnesota Med. *35:* 925 (Oct.) 1952.
19. Jones, T. B.: Personal communication, 1955.
20. Le Fevre, H. W., Jr., and Jacques, T. F.: Multiple polyposis in infant of 4 months, Am. J. Surg. *81:* 90 (Jan.) 1951.
21. Lockhart-Mummery, J. P., and Dukes, C. E.: Familial adenomatosis of colon and rectum, Lancet *237:* 586 (Sept. 9) 1939.
22. Long, C. F., Kolmer, J. A., and Swalm, W. A.: Observations on intestines of rats fed inert materials, J. Lab. & Clin. Med. *20:* 475 (Feb.) 1935.
23. Mayo, C. W., De Weerd, J. H., and Jackman, R. J.: Diffuse familial polyposis of colon, Surg., Gynec. & Obst. *93:* 87 (July) 1951.
24. McCarty, R. T.: Genetic factor of familial disseminated polyposis coli, Am. J. Surg. *86:* 500 (Nov.) 1953.
25. Potts, W. J.: Personal communication, 1955.
26. Radke, R. A.: Amebiasis; some features of disease revealed by study of autopsy material from 96 cases, Gastroenterol. *21:* 525 (Aug.) 1952.
27. ——: Studies in chronic amebiasis; clinical syndrome, Gastroenterol. *25:* 1 (Sept.) 1953.
28. Rankin, F. W., and Webb, J. G.: Surgical treatment of polyposis of colon, South. Surgeon. *14:* 671 (Oct.) 1948.
29. Scarborough, R. A.: Personal communication, 1956.
30. —— and Klein, R. R.: Polypoid lesions of colon and rectum, Am. J. Surg. *76:* 723 (Dec.) 1948.
31. Schantz, G. H.: Multiple polyposis of colon, Gastroenterol. *13:* 430 (Nov.) 1949.
32. Smith, W. G., and Jackman, R. J.: Polyposis of large intestine; preservation of terminal segment, Am. J. Surg. *90:* 751 (Nov.) 1955.
33. Soper, H. W.: Polyposis of colon, Am. J. Med. Sc. *151:* 405 (March) 1916.
34. ——: Discussion of paper by Mayo, C. W., and Wakefield, E. G., J. A. M. A. *107:* 347 (Aug.) 1936.
35. Strohl, E. L., and Pontius, G. V.: Polyposis of colon, Arch. Surg. *58:* 708 (May) 1949.
36. Todd, I.: Discussion of paper by Ravitch, M. M., Ann. Surg. *144:* 764 (Oct.) 1956.
37. Webb, J. G.: Personal communication, 1956.

DUODENAL INJURY DUE TO NONPENETRATING ABDOMINAL TRAUMA

THOMAS B. PATTON, M.D.

Birmingham

Injury of the duodenum due to blunt trauma is supposed, by many, to be a surgical rarity. The frequency, according to some authors, varies between 1 and 10 per cent in all cases of intestinal perforations due to nonpenetrating trauma. In the short space of 23 months, we have seen 4 such patients which prompts this report, and a brief review of the problems involved is presented.

The first such case was reported in 1835 and Guibe[5] reported 135 cases from 1835 to 1910. Seventeen ruptures occurred at the duodenojejunal junction, and there was 1 survival. Of 62 patients who were operated upon in this series, there were only 4 survivals.

ANATOMY

One must consider the duodenum in relation to its anatomic location and its fixed portion in relation to the adjacent structures. Except for the first portion, the duodenum is fixed by fusion between its peritoneum and that of the head of the pancreas to the parietal peritoneum. It is also fixed by its connection with the common bile duct and the pancreatic ducts as well as vessels and nerves. The duodenojejunal angle is fixed by the ligament of Trietz. The right colic flexure and fixed portion of the transverse colon and mesocolon anchor the duodenum still more securely. There is also additional fixation through the adhesion of the greater omentum to the transverse mesocolon and the lateral edge of the margins of the diaphragm[3] (fig. 1).

MECHANISM OF INJURY

It would seem that because of its protected position, the duodenum would not be vulnerable to trauma. The very fixity of this structure renders it unable to escape from the impact of a blow to the abdomen, which if of sufficient force, may crush or avulse the duodenum against the vertebral column. Salisbury[9] believed that a more likely mechanism is the dilatation of the second portion of the duodenum with fluid or gas. When the blow to the abdomen is sustained, the contents of the dilated stomach are forcibly ejected through the pyloric sphincter into the duodenum and perforation is inevitable when pressure within the lumen exceeds the tensile strength of the walls. It is of interest that this point was raised by Dr. Charles Mayo in the discussion of Dr. Kanavel's paper in 1913.[8]

OCCURRENCE

Perusal of the world literature showed a total of some 205 cases through 1953. As would be expected, of these patients only 5 were female. It is seen predominantly in active individuals.

From the Department of Surgery, Medical College of Alabama Birmingham, Alabama.

FIG. 1 Anatomic points of fixation rendering the duodenum an immobile organ

SYMPTOMS

The patient usually has sustained a blow or crushing injury of the abdomen. When seen he complains of pain in the right upper quadrant or epigastrium—increasing in severity with passage of time. He may or may not have been nauseated, and in some cases he will have vomited. The vomitus may contain bright red blood. The patient may also describe a fainting episode and complain of weakness.

Examination of the patient may reveal a contusion of the abdominal wall,

diffuse or severe tenderness in the right upper quadrant or in the epigastrium, or a generalized board-like abdominal rigidity depending on whether or not this is an intra- or retroperitoneal rupture. A steady increase in pulse rate may be noted during the time the patient is being observed.

All of these signs and symptoms will vary depending on the time interval existing from the time of injury to the time of examination. The earlier the patient is seen after injury, the greater the likelihood of confusion in making a diagnosis.

The diagnosis will be made only if this condition is considered. It is well to remember that the trauma, or history of trauma, may appear trivial compared with the symptoms and signs elicited. If there is a contusion visible on the abdominal wall, it is wise to explore the patient without undue delay.

DIAGNOSIS

The following aids in diagnosis may render more easily the duodenum suspect and influence earlier exploration. Following a careful history and physical examination, with treatment of shock if present, a Levin tube is passed to determine the presence of old or recently shed blood in the gastric aspirate, since bleeding from the duodenum will sometimes occur following trauma to this area. Abdominal tap should always be performed, for it may yield bile, blood or "beef broth" fluid suggestive of associated pancreatic injury. A negative tap does not exclude the diagnosis, and a positive tap done after the injury should suggest the diagnosis of intraperitoneal rupture. The tap, if negative initially, should be repeated in 4 hours.

Roentgenograms in both the flat and upright positions may reveal retroperitoneal air which may appear as grape-like clusters of rounded shadows about the duodenum, or may dissect downward to the right perinephric area and along the psoas muscle.[11] If the roentgenograms are negative and operation is deferred, these films should be repeated at 4 hour intervals (Case 3).

Air may extend into the pelvis and may be felt on rectal examination.[2] The presence of retroperitoneal air may also give rise to testicular pain.[2] Symptoms and signs may predominate in the right lower quadrant due to the retroperitoneal spread of bile, gastric contents and pancreatic ferments.

TREATMENT

The Levin tube should be left in place to remove as much gastric secretion as possible to prevent the activation of pancreatic ferments. The adjunctive use of Banthine or Atropine by the parenteral route is suggested. Associated pancreatitis with duodenal injury caused the death of 2 patients whose cases were reported by Dixon.[4]

Exploration should be performed promptly when any of the previously mentioned signs are present. When the abdomen is opened, one may find a "clean" abdomen. If laceration or rupture of the peritoneum has occurred along with rupture of the duodenum, bile stained fluid, "beef-broth" fluid or blood may be seen. In the so-called clean abdomen, one must be careful to palpate for crepitus

about the hepatic flexure of the colon and over the duodenum, since this area may appear perfectly normal on inspection. This, indeed, may be the only clue to underlying injury to the duodenum in a patient that is explored early after injury.

The duodenum should always be directly inspected. A high index of suspicion may be entertained if there is any bile stained discoloration over the duodenum or hepatic flexure area (LaFitte's tache verte),[7] since this is pathognomonic of extraperitoneal rupture of the duodenum. In all patients the duodenum must be mobilized. Kocher's maneuver will serve well for the first and second portions of the duodenum, but exposes the third portion poorly. We believe it would be well to quote Kanavel in his own words: "I, therefore, turned the transverse colon and omentum upwards . . . this brought into the field the transverse duodenum, hepatic flexure of the colon and the root of the mesentery. Incision of the peritoneum between the reflected hepatic colon and the duodenum freely exposed the third portion of the duodenum."[6] It is important to thoroughly visualize the entire posterior aspect of the duodenum. Unless this is done, an adequate exploration has not been carried out. These rents are easy to miss; for Berry and Giuseppi[1] report that out of 13 patients explored, only 7 ruptures were discovered, 6 being found at postmortem, most of which were extraperitoneal.

If a rent is found in the duodenum, it is closed in the usual fashion, using a Connell suture for the first layer and placing a second layer of interrupted cotton sutures. Drains are placed near, but not on, the area of repair, and the duodenum allowed to fall back in its normal position but is not sutured. Antibiotics are administered.

CASE REPORTS*

Case 1: C. W., was a 37 year old Negro man. This man was helping carry a heavy cabinet up the stairs of a house at 5:30 p.m., on January 6. The man assisting him, and above him on the stairs, slipped and the cabinet fell from his grasp. The corner of the cabinet struck the patient in the abdomen and pinned him between the stairwell and the cabinet. He immediately experienced severe epigastric pain and vomited red blood. He was seen by a physician who urged him to enter the hospital, but the patient refused and returned home. He was seen in the Admitting Room on the following day, January 7, and was admitted to the hospital at 2:15 p.m.

Examination revealed a man in shock. His blood pressure was 80/50 and he complained of severe generalized abdominal pain. His abdomen was board-like and no peristalsis could be heard. There was marked tenderness on rectal examination. His white blood cell count was 14,450 per cu. mm., with 89 per cent polymorphonuclear leukocytes. Roentgenograms of the abdomen revealed some dilatation of bowel loops; and in the region of the hepatic flexure, there was a spongy, gaseous shadow which was interpreted as being suggestive, but not diagnostic, or retroperitoneal air. A diagnosis of duodenal rupture was made and the patient was taken to the operating room at 7:10 p.m., 26 hours after injury.

He was explored through a right subcostal incision. There was bile tinged fluid in the peritoneal cavity and a rent approximately ¾ inch in length was found in the descending portion of the duodenum beneath the leaves of the transverse mesocolon. This rent was closed in two layers and reinforced with omentum. The abdomen was closed without drainage.

* Service of Dr. C. G. Johnston, Detroit Receiving Hospital, Detroit.

The patient had an exceedingly stormy postoperative course and died at 10:55 a.m., the day following laparatomy. No postmortem examination was permitted, and it was concluded that he died of retroperitoneal cellulitis and irreversible shock.

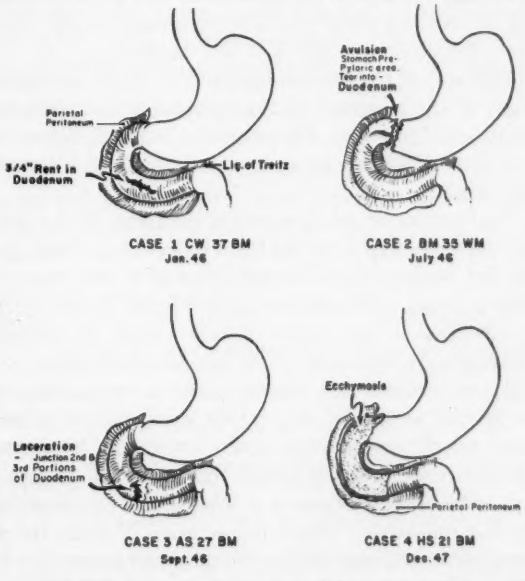
Case 2: B. M., was a 35 year old white man. This patient was caught between a truck and a loading platform at approximately 10:00 p.m., on July 11. He was brought to the Admitting Room in shock and complaining of severe abdominal pain. He was treated for shock and roentgenologic examination revealed free air beneath the diaphragm. He was taken to the Operating Room and explored through a left subcostal incision.

There was complete avulsion of the stomach in the prepyloric area and an associated tear into the first portion of the duodenum. A subtotal gastric resection was performed and the patient recovered without incident. This patient was operated upon 3 hours after the injury. Ten months later he perforated a marginal ulcer.

Case 3: A. S., was a 37 year old Negro man. This man was injured on September 6 at 9:30 a.m., when he fell from a scaffold one story in height. At the moment of the impact with the ground, the air hammer which he was using struck him in the abdomen. He was immediately brought to the hospital.

When seen in the Admitting Room, he was vomiting, writhing in pain and complaining of severe epigastric pain. At this time, his pulse was 78 and his blood pressure was 158/60. The abdomen was flat and there was a 4 by 4 centimeter contusion over the epigastrium where there was tenderness and rebound tenderness. No bowel sounds were heard on auscultation. A Levin tube was passed. Aspirate from the stomach revealed no blood. The patient was transferred to the ward. Continuous suction was applied to the Levin tube and abdominal paracentesis performed on the right side. Air and material resembling feces was obtained and the operator thought that he had entered the bowel. At this time, his abdomen was more soft and he said he was more comfortable.

Roentgenograms taken on admission did not reveal any abnormality, but repeat films made later in the day, revealed small vesicles of retroperitoneal air adjacent to the duo



DUODENAL INJURY DUE TO NON PENETRATING ABDOMINAL TRAUMA

FIG. 2 Graphic presentation of the 4 cases discussed

denum and in the right perirenal space. A diagnosis of ruptured duodenum was made and he was taken to the operating room.

Exploration of the abdomen was performed through a right subcostal incision, and there was bile staining over the duodenum. The peritoneum was intact. The duodenum was mobilized and there was almost a complete division of the duodenum at the junction of the second and third portions. This laceration was repaired in two layers and the abdomen drained. The patient had a stormy postoperative course and gradually recovered. The time interval between injury and operation was 12 hours.

Case 4: H. S., was a 21 year old Negro man. This man fell a distance of 3 to 4 feet from the back of a truck from which he was alighting and struck his epigastric area. This occurred at 8:00 a.m., December 23. At 10:00 p.m., of the same day, he was nauseated and vomited twice before presenting himself to the Admitting Room of the hospital at 3:45 a.m., December 24. Examination revealed a board-like abdomen, with absent bowel sounds. His blood pressure was 130/70 and his pulse 100. Roentgenograms of the chest and scout films of the abdomen did not reveal any abnormality. He was transferred to the ward, and a Levin tube passed and no blood was seen in the aspirate obtained. Despite the negative findings, the patient was taken to the operating room and explored because of abdominal rigidity and absent bowel sounds. A right subcostal incision was made because of maximal tenderness in the quadrant of the abdomen.

There was marked extraperitoneal ecchymosis completely obscuring the duodenum, and because it was feared that the blood supply to the duodenum had been compromised, a duodenectomy was performed. Convalescence was satisfactory except for a wound infection. Microscopic examination of the duodenum revealed marked submucosal hemorrhage. This patient was operated upon 25 hours after his accident (fig. 2).

DISCUSSION

It is apparent that any trauma to the abdominal wall which is nonpenetrating but of a squeezing or crushing variety may cause a catastrophic series of events if there is contusion, laceration or rupture of the duodenum.

Intraperitoneal ruptures of the duodenum are more easily recognized as acute emergencies because of the clearness of the symptoms and signs than are the extraperitoneal ruptures. Moreover, the prognosis is much better in intraperitoneal ruptures, for there is less chance for retroperitoneal cellulitis and associated pancreatitis to develop.

It is apparent that extra- or retroperitoneal ruptures of the duodenum are analogous to spontaneous extrapleural ruptures of the lower third of the esophagus. The urgency for surgical intervention is present and recognized, but a definitive diagnosis is much more obscure and difficult. In the early period of duodenal injury, cellulitis of the retroperitoneal tissues is advancing just as mediastinitis is developing in the case of the extrapleural rupture of the esophagus. Indeed, confirmatory signs may appear earlier in spontaneous perforation of the esophagus invested as it is by only a thin layer of parietal pleura; for one will remember that two layers of peritoneum invest the duodenum and considerable inflammation may progress before this structure gives way. Perhaps the greatest aid in these difficult cases is a careful physical examination of the patient, not once, but repeatedly. The demonstration of air in the periduodenal area or the perirenal area, although it may be minimal, if detected early, should encourage careful scrutiny of the abdominal films and will enable an accurate diagnosis.

Delay is fatal and the interests of the patient will best be served by exploration where the diagnosis is suspected. One should not forget the information which may be obtained by the injection of radiopaque materials through the Levin tube which may actually pass into the retroperitoneal area and prove the diagnosis.[10] If operation is delayed 24 or 48 hours, until definite diagnostic features are present, the chances of a positive diagnosis will be more accurate, but the chances of the patient's survival are diminished.

In 3 of the patients presented, the trauma was not minimal, but it was difficult to diagnose the sequence of events occurring in the abdomen. If there is no retroperitoneal air demonstrable on the abdominal films, and if there is no bile stained or "beef broth" fluid obtained on abdominal tap, early exploration may be hard to justify. If, however, there is a mark of contusion, ecchymosis or hematoma of the abdominal wall following a history of trauma, we believe that exploration is justified forthwith.

CONCLUSIONS

Four cases of retroperitoneal laceration and contusion of the duodenum have been presented.

Anatomic features rendering the duodenum peculiarly vulnerable to nonpenetrating trauma of the abdominal wall have been reviewed.

Diagnostic features which may aid in the establishment of accurate preoperative diagnosis are re-emphasized.

Early exploration with careful inspection, palpation and mobilization of the duodenum will markedly reduce morbidity and mortality from this lesion.

Any evidence of trauma to the abdominal wall is sufficient evidence of significant intraperitoneal trauma to make early operation mandatory.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Berry, J., and Guiseppi, P. L.: Traumatic rupture of intestine, with case of recovery after operation and analysis of 132 cases that have occurred in 10 London Hospitals during last 15 years (1893-1907), Proc. Royal Soc. Med. Vol. II, Part III, Surgical Section, London, Oct. 13, 1908.
2. Butler, E., and Carlson, E.: Pain in testicle, symptoms of retroperitoneal traumatic rupture duodenum, Am. J. Surg. *11:* 118 (Jan.) 1931.
3. Callander, C. L.: Surgical Anatomy, 2nd ed., Philadelphia, W. B. Saunders Co., p. 332, 1939.
4. Dixon, G. G.: Duodenal rupture and fat necrosis; report of 2 cases, Ann. Surg. *114:* 147 (July) 1941.
5. Guibe, M.: Contusions et ruptures traumatiques du duodenum, Rev. de Gynec. et de chir. Abd. *15:* 223, 349, 1910.
6. Kanavel, A. B.: Duodenum, mobilization, traumatic rupture and toxemia, Tr. West. Surg. Assoc. p. 221, 1913–1914.
7. Lafitte, H.: Contribution a l'etude des ruptures sous peritoneales due duodenum (Valeur de la tache vert), Arch. franco-belges de chir. *34:* 117 (Feb.) 1934.
8. Mayo, C.: Discussion, Tr. West. Surg. Assoc., p. 234, 1913–1914.
9. Salisbury, C. V.: Traumatic rupture of second part of duodenum, Lancet *2:* 563 (Nov. 3) 1945.
10. Siler, V. E.: Management of rupture of duodenum due to violence, Am. J. Surg. *78:* 715 (Nov.) 1949.
11. Sperling, L., and Rigler, L. G.: Traumatic retroperitoneal rupture duodenum; description of valuable roentgen observation in its recognition, Radiology *29:* 521 (Nov.) 1937.

THE VALUE OF EMPLOYMENT OF THE COMPONENT PARTS OF THE TRANSVERSALIS FASCIA AND COOPER'S LIGAMENT IN THE REPAIR OF THE LARGE OBLIQUE, DIRECT AND RECURRENT INGUINAL HERNIA

DAN C. DONALD, M.D.

Birmingham

Formerly the Bassini operation, or one of its modifications, was employed in which the various deep layers of the inguinal space were sutured to the inguinal ligament for the large oblique, direct and recurring hernia. Recurrences were frequent, particularly in the direct hernia, where the rate ran upward of 10 per cent to 15 per cent. Our recurrences following the above type of repair were a stimulus for seeking a better understanding of the inguinal hernia problems.

The personal and referred recurrent patients who had had their hernias repaired by the conventional procedure were found to have a definite weakness of deep inguinal support regardless of whether the Bassini, the Halsted, or the Ferguson technique had been employed.

The points of weakness were; first, the defect in the transversalis fascia responsible for the primary hernia had not been closed. Second, instead of closing the defect in the deep inguinal space, support for these tissues was made by bridging or approximation of the conjoined tendon and the medial layer of the transversalis fascia (Hesselbach's ligament) to the inguinal ligament over the transversalis fascia defect. In no patients were there indications that the outer segment of the transversalis fascia (iliopubic tract) had been included in such approximation.

Anatomically and physiologically such a procedure is unsound for the following reasons: first, there is failure to close the hernia defect in the transversalis fascia by the approximation of its two opposing segments (Hesselbach's ligament and the iliopubic tract). Second, the approximation of tissues of different structure, such as the conjoined tendon and Hesselbach's ligament (consisting of muscle and fibrous tissue) to the inguinal ligament, (composed of fascia tissue). Third and finally, closure of the deep inguinal space above the pubic bone in the large direct hernia will often place the sutures under strain or tension sufficient to interfere with normal tissue repair.

For the past 16 years utilization of the component parts of the transversalis fascia and Cooper's ligament has been employed in all of the large oblique, direct and recurrent inguinal hernia patients. In children and young adults who do not show damage to the transversalis fascia, the Bassini operation is employed. In this latter group of patients, the hernia is predominantly sac formation with varying degrees of dilatation of the internal abdominal ring.

A preliminary report of our results extending over an 8 year period has been published previously. There were 98 patients, 18 of whom had bilateral hernias,

From the Department of Surgery, Medical College of Alabama, Birmingham, Alabama.

giving a total of 116 hernia operations. A questionnaire was sent to each patient asking if there had been any recurrence of their hernia. There were replies from all but 3 patients whose questionnaires were returned as unclaimed. In the answered questionnaires, without exception, the patient stated that there had been no recurrence.

During the past 8 years from Jan. 1, 1947 through Dec. 31, 1955, the total number of hernia operations was 248. There were 204 patients, of whom 44 had bilateral hernias. During this 16 year period from Jan. 1, 1939 through Dec. 31, 1955, 364 hernia operations were done. All were private patients. Forty-five of the patients or 15.1 per cent were children and young adults, whose ages ranged from 2½ to 30 years. It was in this latter group that the Bassini operation was employed. Of 248 hernia operations, 136 were for oblique hernias; 87 for direct and 25 for recurrent. A questionnaire was sent recently to each patient who was operated upon during this last 8 year period, from Jan. 1, 1947 through Dec. 31, 1955, asking if there was any recurrence of hernia. There were replies from all except 5 patients. In the answered questionnaires, two recurrent hernias were reported. One of these was in a child 14 years of age, who was reported to have had a recurrence of the hernia 6 months after operation. On contact with the surgeon in charge of the patient, it was found that this patient had a femoral hernia and not a recurrence of the oblique hernia repaired by us. Evidently the femoral hernia was present and overlooked at the time of the inguinal hernia repair.

The second patient with hernia recurrence whose case was reported was a male 45 years of age. Upon examination requested by us of the inguinal area, we found the hernia to consist of two small hernia defects, independent of one another, just above the pubic bone. Most likely this resulted from giving away of the repaired tissues due to tension on the suture line at the base of Cooper's ligament and the component parts of the transversalis support.

THE ANATOMY OF THE DEEP INGUINAL SPACE

The anatomy of the deep inguinal space comprises chiefly the transversalis fascia, superimposed by the internal oblique and transversus abdominis muscles medially, the inguinal ligament laterally, and the aponeurosis of the external oblique muscle. The transversalis fascia which is the principal supportive structure in the inguinal region is normally weakened in its upper portion by the internal abdominal ring, which provides an exit for the passage of the spermatic cord in the male and the round ligament in the female.

The second point of weakness of the transversalis fascia is in the lower inguinal space commonly called Hesselbach's area where it does not receive much support from the internal oblique and transversus abdominis muscles.

It is through these two respective areas that all inguinal hernias occur. To obtain the maximum results from surgery for the correction of inguinal hernia it is essential to have a full knowledge of the transversalis fascia.

In the inguinal region the transversalis fascia furnishes the chief fascial support of the anterior abdominal wall. Here the fascia becomes thick and strong, re-

inforcing the unprotected areas. Its fibers assume a fan shape as they receive support at their different points of insertion onto the pubic bone and the different ligamentous tissues of this area. Along its medial portion it becomes blended with the fascia of the rectus muscle and laterally to the dense iliopsoas fascia beneath the inguinal ligament.

From the lower inguinal region it projects into the thigh with the femoral vessels as the femoral sheath. As a result of its modifications in the inguinal region the following component parts of the transversalis fascia are derived: the ligament of Hesselbach (ligament of interfoveolare), Henle's ligament, and the iliopubic tract.

These respective structures blend at the pecten of the pubes to form Cooper's ligament. Cooper's ligament extends from the tubercle of the pubes along its superior border to the ilio pectineal eminence. It appears as a thick fibrous cord intimately adherent to the periosteum of the pubic bone and runs posteriorly at an angle of 30 to 35 degrees with the inguinal ligament.

Because of its strength and position, its utilization in the repair of large, oblique, direct, and recurrent inguinal hernias is very important. A detailed

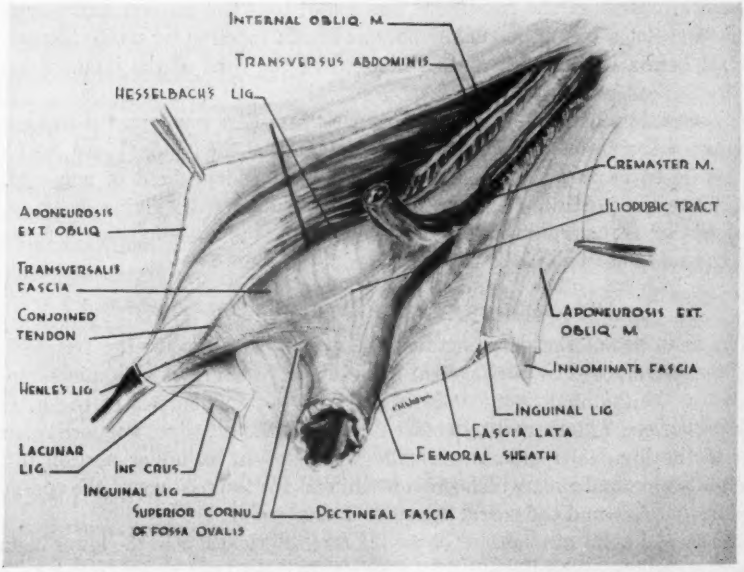


FIG. 1. The transversalis fascia in the inguinal region. The relationship of the iliopubic tract, transversalis fascia, and the femoral sheath is shown. The inguinal ligament and fascia lata have been cut and reflected. The transected muscular portions of the internal oblique and transversus abdominis muscles are reflected to show the lateral portion of the transversalis fascia as it is continuous with the femoral sheath below. The separation of the conjoined tendon into its component parts (the transversus abdominis and internal oblique muscles) is shown. Henle's ligament is an inferior lateral expansion of the enveloping fascia and tendon of the rectus muscle, blending and continuous with the iliopubic tract and femoral sheath.

anatomy of the transversalis fascia, its component parts and Cooper's ligament is presented by the accompanying drawings: figures 1, 2, 3.

Anatomically the inguinal ligament is weak in the low inguinal region. The only support it receives is from its insertion to the pubic bone medially, the anterior superior iliac spine laterally, and its attachment below to the fascia lata. Surgically, the inguinal ligament does not provide a suitable substitute for the insertion of these structures (transversalis fascia, transversus abdominis, and the internal oblique muscles). Its relationship to these structures is merely one of proximity.

HISTORIC

Prior to the past three or four decades not much thought was given to the relationship between hernia formation and the defective transversalis fascia.

The disappointment in the surgical results following hernia repair by the routine use of the inguinal ligament led to an awakening of surgeons as to the value of the employment of Cooper's ligament.

In 1804 Ashley Cooper[4] described the ligament which bears his name and suggested it might be used in the repair of femoral hernia. In 1876 Annadale[1] used Cooper's ligament in repairing a femoral hernia.

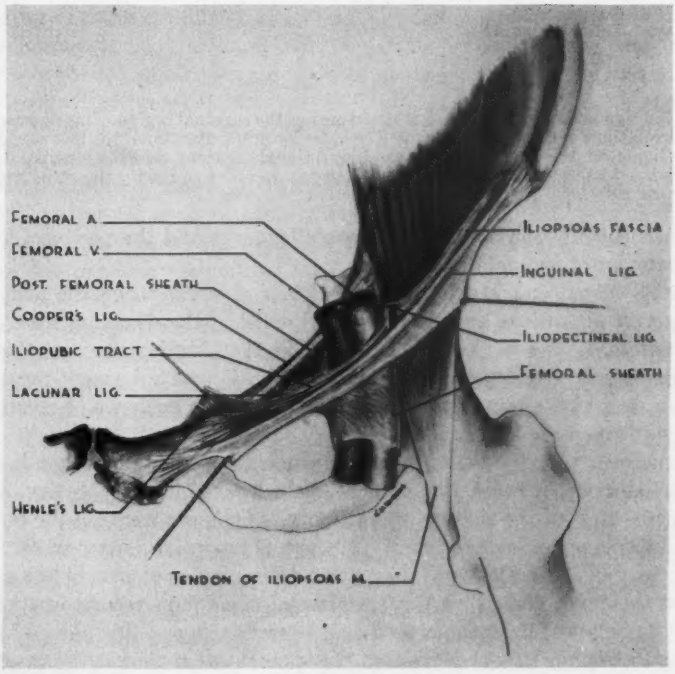


FIG. 2. This diagram shows the location of Cooper's ligament, attached to the pubic bone inferiorly and to the iliopectineal eminence posteriorly, and its relationship to the inguinal lacunar ligaments. The weak support of the inguinal ligament is apparent.

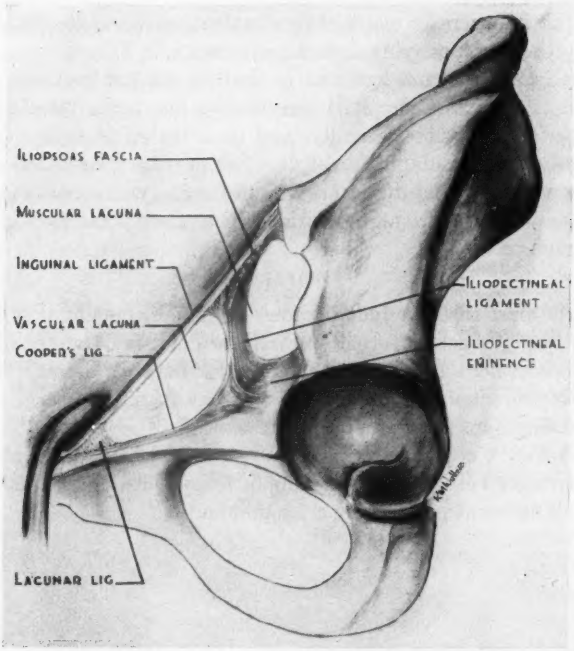


FIG. 3. Deep bony and ligamentous structures of the inguinal region. This diagram shows the interrelations of Cooper's ligament (the superior pubic ligament), the lacunar ligament, Henle's ligament, the iliopubic tract, the iliopectineal ligament, and the inguinal ligament. The femoral vessels are displaced slightly laterally in order to clarify the structures about the femoral canal.

Lotheissen[10] in 1898 employed Cooper's ligament and the pectineal fascia in the treatment of femoral hernia.

In 1938 Anson and McVay[12] reported their observation on the anatomy of 125 cadaver dissections and later McVay published the details of his operative procedure in the use of Cooper's ligament. In 1949 McVay reported the results of operations on 100 patients, 56 of whom had a Cooper's ligament repair. With a follow up period of up to 8 years, McVay found no evidence of recurrence in his 56 patients although 4 were lost in the follow up period.

Zimmerman[15] in discussing inguinal ligament repair, stated that hernia recurrence does occur below the inguinal ligament as stated by McVay. Zimmerman's criticisms of the routine use of Cooper's ligament were (1) the repair is a more difficult operative procedure, (2) there is potential danger to the femoral vessels as the tissue layers are sewn down to the ligament plus creating a weak area at this point and (3) at this transitional angle most recurrences will occur unless the inguinal ligament is used as a lateral anchor in this phase of the procedure. Burton[3] in 1954 thought that the transitional angle was the danger point in McVay's repair. In 1949 Harkins[7] reported the results of 398 cases of Cooper's method, with 3 recurrences (0.8 per cent). Mayo and Keeley,[11] employing a

modification of McArthur's method for a period of 12½ years, operated on 222 patients. One hundred and fifty-five cases were unilateral, with a recurrence rate of 11.9 per cent and 34 cases were bilateral, with a recurrence of 13 per cent. In both groups this percentage of recurrence was for 5 years observation.

Bowers and Schear,[2] analyzed the statistics from 18 military hospitals for 6 years, Jan. 1, 1946 through Dec. 31, 1952. Of 5834 hernia operations, in which the majority of repairs were the conventional procedure employing the inguinal ligament, and a small percentage employing Cooper's ligament, there were 482 recurrences, (8.3%).

Estes[5] in 1956, using a modified Cooper's method, reported 363 cases with 5 recurrences, (1.5 per cent).

The hazards to avoid in hernia repair are the surgeon's errors viz: (1) improper removal of hernia sac, (2) failure to search for additional hernias, (3) undue trauma to the tissues, (4) nerve injury, (5) careless hemostasis, (6) proper skin preparation, and (7) improper suture material (nonabsorbent material such as cotton or silk is preferred).

SURGERY

For anesthesia, nitrous oxide and oxygen and spinal anesthetic for adults; open drop ether for children.

The Bassini incision is employed. The incision should not extend onto the scrotum or the low pubic area, as the skin in these areas harbors bacteria and if these tissues are incised it may lead to infection of the wound. In the only 2 cases of infection in this series, both occurred where incisions had been extended onto these areas. With a blunt retractor placed in the lower angle of the wound, sufficient exposure of the deeper structures can be obtained without incising the low pubic or scrotal area. In cases of hernia the superficial vessels of the lower angle of the wound often are larger and more numerous than in the upper angle, possibly due to the irritation from the hernial bulge or pressure from a truss. To maintain hemostasis the larger vessels should be carefully dissected and doubly clamped before they are divided. The smaller vessels and capillaries should be clamped and ligated. This prevents hematoma and minimizes serum accumulation, thus lessening the danger of infection and interference with tissue repair.

Suture material should be either cotton or silk. A heavy grade is used for the deep sutures, medium for the external aponeurosis and Scarpa's fascia, and fine for hemostasis. The chief value of nonabsorbable over absorbable sutures (catgut) is that they maintain tissue apposition until union occurs, and tissue reaction for serum accumulation is lessened further reducing the risk of infection.

The skin incision is carried through Scarpa's fascia to the aponeurosis of the external oblique muscle, where sponge dissection should be employed to clear away the areolar and adipose tissues from the surface of the aponeurosis throughout the incision. The aponeurosis is opened by a small incision in the upper angle of the wound. With elevation of the margins of the aponeurosis, the incision is carried through the fibers down to and including the subcutaneous ring. The ring is opened along its upper medial surface, bringing the ilioinguinal and iliohypo-

gastric nerves into view. The ilioinguinal nerve runs parallel to and medial with the spermatic cord. Opposite the subcutaneous ring the ilioinguinal nerve divides into several branches, some of which often pierce the ring fibers of the external oblique aponeurosis. Good exposure prevents injury to these nerve fibers when the ring is opened. The iliohypogastric nerve makes its superficial exit several centimeters above the bed of the ilioinguinal nerve. It runs in a transverse direction and pierces the rectus fascia along its outer margin. Injury to either nerve may result in weakening the muscles, which may encourage a recurrence of the hernia. Also, injury to the ilioinguinal nerve and its fibers at the subcutaneous ring will produce a numbness of the scrotum and medial skin surface of the thigh.

The ilioinguinal nerve should be protected by being dissected from its bed and withdrawn in an outward lateral manner. The spermatic cord is separated from its anatomic bed and withdrawn in a like manner. In many patients the hernial sac is of such size that it fills the entire canal. It is difficult in such patients to determine whether the hernia began as an oblique or a direct type, since there is loss of support of the entire transversalis fascia. The deep epigastric vessels which normally run inferior and medially to the internal abdominal ring no longer serve as a guide for diagnosis between an oblique and a direct hernia. When the hernial bulge attains large size these vessels are sometimes displaced to a position above and opposite the internal abdominal ring. The diagnosis of small direct or femoral hernia suggested by Fallis[6] constitutes a real advance in the recognition of these hernias when they have not been observed by routine examination. In patients with a small direct hernia Fallis suggested opening the peritoneum at the internal abdominal ring above and medial to the spermatic cord. The surgeon's finger then enters the peritoneal cavity and the entire inguinal space, including the femoral region, is palpated for weak points and peritoneal pouching.

In the management of small direct hernias, or where the dome portion of the transversalis fascia is herniated, and when they accompany oblique hernia, their treatment should be carried out in the fashion recommended by Hoguet.[9] However, for the large direct hernia with definite sac formation, the Hoguet method is not applicable. This type of hernia is treated in the same way as an oblique hernia. This is accomplished by freeing the hernial sac of adhesions down to the abutment of the peritoneum. In both large direct and oblique hernias, the hernial opening in the peritoneum is closed by a purse-string suture placed on the inside of the sac at its base. An alternate for the purse-string suture is the transfixed stitch placed at the base of the open sac. In the large direct hernia, the hernial ring in the transversalis fascia must be closed independently of the reconstruction of the fascia.

The practice of anchoring the stump of the sac to the undersurface of the overlying muscles in the oblique hernia has been advocated by some as the best method of preventing recurrence of hernia. It has been observed that this treatment may lead to the pocketing of the peritoneum at the point of anchorage and invite recurrence. Passing the suture through and over the muscles may also interfere with the action of the muscles and weaken them by strangulation.

The complete removal of the hernial sac and closure of the internal abdominal ring are essential for a successful operation for oblique hernia. In the closure of the ring both arms and the angle of the ring should be identified. This can be done by elevating the spermatic cord and freeing adhesions beneath the cord by sponge dissection down to the angle of the ring. The angle of the ring will appear as dense fibers firmly adherent to the bed of the transversalis fascia. From this point each arm of the ring will be seen to run in line with its normal position. To facilitate the exposure of each arm, an Allis clamp may be placed on its fibers, and with elevation of the clamp the respective arms will come into view (fig. 4). After such exposure the arms of the ring are approximated by 2 to 3 interrupted sutures above the cord. In placing the sutures one should avoid incorporating the spermatic cord tissues or closing the ring so tightly as to constrict the cord.

After the hernial sac and the hernial ring have been properly treated, it is necessary to clear the inguinal canal of areolar and adipose tissue, accessory

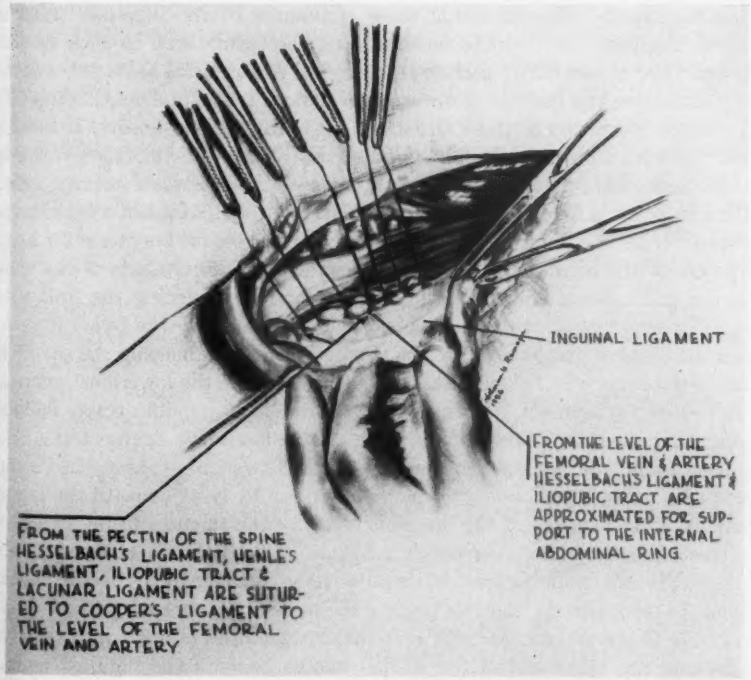

INGUINAL LIGAMENT
FROM THE LEVEL OF THE FEMORAL VEIN & ARTERY HESSELBACH'S LIGAMENT & ILIOPUBIC TRACT ARE APPROXIMATED FOR SUPPORT TO THE INTERNAL ABDOMINAL RING
FROM THE PECTIN OF THE SPINE HESSELBACH'S LIGAMENT, HENLE'S LIGAMENT, ILIOPUBIC TRACT & LACUNAR LIGAMENT ARE SUTURED TO COOPER'S LIGAMENT TO THE LEVEL OF THE FEMORAL VEIN AND ARTERY

FIG. 4. The defect of the transversalis fascia through which the hernia of the lower inguinal space occurs has been corrected by the approximation of the component parts of the transversalis fascia (the ligaments of Hesselbach, Henle, Lacunar and iliopubic tract) to the Cooper's ligament up to the level of the femoral vessels. From this point to the internal abdominal ring, the support of the deep inguinal space is made by the approximation of the Hesselbach ligament to the iliopubic tract. The figure shows the sutures in place. For the correction of the dilated internal abdominal ring, the medial and outer arm of the ring have been identified and brought into view by Allis clamps at each arm preparatory for approximation of the arm of the internal ring above the cord.

vessels, and straggling muscle fibers. Most frequently the accessory vessels are found beneath the cord as it swings over the pubic bone. Often the vessels are of large size and they should be doubly clamped before dividing. In the recurrent hernia varying fibrous bands of tissue will be found adherent to the inguinal ligament from the previous approximation of the supportive structures to the ligament. The inguinal ligament is to be freed of the fibrous tissue. This affords good visualization of the operative field, including the component parts of the transversalis fascia and Cooper's ligament. Cooper's ligament may be identified as a fibrous cord by palpation of the superior margin of the pubic bone. It is covered by the thin pectineal fascia, and it can be readily discerned by placing an Allis clamp onto the pectineal fascia so as to include the ligament.

For statistical purposes the size of the hernial cleft may be obtained by a linear and transverse measurement of the cleft, using a graduated uterine probe.

Upon examination, the hernial cleft may be found to be of such size that, with retraction of the Hesselbach ligament and the aponeurosis of the transversus abdominis muscle, approximating these structures to the iliopubic tract and Cooper's ligament may lead to tension along the suture line. In such patients, Rienhoff[13] and others have suggested incising the rectus fascia at its pubic attachment, extending the incision in an upward manner for several centimeters. This will provide relaxation of these structures for normal tissue closure. It has been found that such a procedure will not weaken the rectus muscle and the pyramidalis muscle support. However, in only one patient in this series was a relaxing incision in the rectus fascia found necessary, due to atrophy of Hesselbach's ligament and the transversus abdominis aponeurosis following a previous operation for hernia.

Closure of the hernial cleft should be begun at the lower angle of the wound over the pubic bone. Harkins and Swenson[8] preferred placing the first suture several centimeters above the pubic bone in proximity to the femoral vein to lessen the chance of injury to the vein. I recommend commencing the suture line over the pubic bone and passing the first suture through the lowermost portion of of Hesselbach's ligament, Henle's ligament, and the iliopubic tract, including Cooper's ligament and fibers of the lacunar (Gimbernat's) ligament (fig. 4). It has been found that this procedure accomplishes two things: first, the iliopubic tract at its pubic insertion is often small and obscure, lying beneath the inguinal ligament. The elevation of the iliopubic tract by the suture will better identify the tract. Second, there is a better coaptation of the ligamentous structures. Occasionally, the iliopubic tract at its pubic insertion may be so obscure that it is difficult to recognize its fibers for placing the first suture. In such patients, before the suture line is commenced, the iliopubic tract can be brought into clear view by passing the blunt end of the scalpel handle beneath the inguinal ligament opposite the pubic bone to separate the loose adhesions between the inguinal ligament and the iliopubic tract. In this way the aponeurotic fibers of the iliopubic tract will be clearly differentiated from the white fibers of the inguinal ligament. Utilizing the iliopubic tract gives essential lateral support to the transversalis fascia.

The sutures are placed 1 to 2 cm. apart, and upon placement of the third

suture, which is the last one incorporating Cooper's ligament, attention is given to the femoral vessels (fig. 4). To avoid injury to these vessels it is necessary for the surgeon to place his index finger into the wound and retract the vessels in a lateral manner before the suture is inserted. The suture line for approximating Hesselbach's ligament and the transversus abdominis muscle to the iliopubic tract is continued in an upward manner to the level of the internal abdominal ring (fig. 4).

Testut and Jacob,[14] in their anatomic description of the iliopubic tract, and McVay and Anson,[12] discussing the clinical use of the iliopubic tract, have spoken of the diminution in the size of the tract as it crosses the femoral vessels (figs. 2, 3). It has been found, however, that the iliopubic tract is not altered at this point to such an extent as to interfere with approximation of the tract to Hesselbach's ligament and the transversus abdominis aponeurosis.

The operation is completed as follows: the spermatic cord and the ilioinguinal nerve are replaced into the inguinal bed and permitted to rest on the suture line following the closure of the transversalis fascia (fig. 5). The open or dead space above the pubic bone, bounded laterally by the spermatic cord and the inguinal ligament, medially by the rectus fascia below, and above and medially by the internal oblique and transversus abdominis muscles, is dealt with in the following manner: first, if the case is one in which there is a congenital absence of the internal oblique and transversus abdominis muscles in the low inguinal space, or atrophy of these muscles from disuse, nutritional deficiency, or following necrosis secondary to previous surgery, no attempt is made to close the space except to loosely approximate the muscle fibers of the internal oblique and transverse abdominis muscles to the inguinal ligament, above the cord, opposite

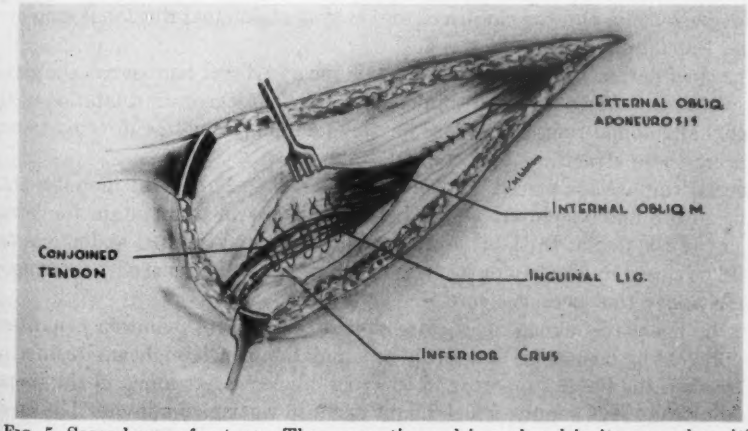


FIG. 5. Second row of sutures. The spermatic cord is replaced in its normal position, resting on the first row of sutures. The fibers of the conjoined tendon are loosely approximated to the under surface of aponeurosis above the cord. The extent of the approximation of the conjoined tendon to the inguinal ligament depends on the condition of the muscle fibers in the low inguinal region (see text). Shown also is the beginning of the closure of the external oblique aponeurosis, with interrupted sutures.

the internal abdominal ring. Second, if the internal oblique and transversus abdominis muscle fibers are healthy and well developed, they are loosely approximated to the inguinal ligament, above the cord, opposite the internal abdominal ring as far downward as these muscle fibers extend. In closure of the fibers of the external oblique aponeurosis and the reconstruction of the subcutaneous ring, care should be taken to avoid constriction of the spermatic cord by sutures (fig. 5). Closure of Scarpa's fascia and the skin completes the operation.

SUMMARY

During a 16 year period from Jan. 1, 1939 through Dec. 31, 1955, 364 hernia operations were done. Forty-five of the patients or 15.1 per cent were children and young adults whose ages ranged from 2½ to 30 years. For the latter group the Bassini operation was employed, in the 319 remaining patients with large oblique, recurrent and direct hernias in whom this procedure was employed, questionnaires sent to the patients showed only 1 recurrence of hernia, with all questionnaires answered except 8, which were returned unclaimed.

To further the knowledge of the deep supportive structures of the inguinal region, an anatomic review has been made of these structures. This has consisted of a review of the literature in this field, cadaver dissections, autopsies, and clinical observations at operations.

The pathology of all inguinal hernias is not the same. To obtain the maximum results in the operation for inguinal hernia, it is necessary to consider the pathologic changes that accompany the different types of hernias in the inguinal supportive tissues. Since the transversalis fascial support is not involved in the small oblique hernia, usually seen in children, the treatment consists simply of the removal and closure of the patent processus vaginalis (sac) down to the fold of the peritoneum and the closure of the internal abdominal ring, as is employed in the Bassini operation.

The chief pathologic features in large oblique and direct hernias are the giving way of the transversalis fascial support and varying degrees of dilatation of the internal abdominal ring. Operations for these hernias should be directed toward restoring these structures to a normal state.

Special emphasis is placed on the importance of including the iliopubic tract in the suture line for closure of the transversalis fascia to maintain the lateral support of the fascia. In the oblique hernia, the dilated internal abdominal ring should be closed by identifying its arms and approximating them with interrupted sutures above the spermatic cord.

In the operative management, care should be taken to maintain hemostasis, thus preventing hematoma and serum accumulation which might invite infection and weaken the tissue support, and to avoid unnecessary trauma of the tissues, including the nerve supply, which might result in weakened muscles. The operative field should be cleared of areolar and adipose tissues and straggling muscle fibers for good visualization and prevention of their incorporation in the suture line, which would weaken the ligamentous support.

Department of Surgery
Medical College of Alabama

REFERENCES

1. Annadale, L.: Case in which a reducible oblique and direct inguinal hernia and femoral hernia existed on the same side, successfully treated by operation, Edinburgh, M. J. 1087, 1876.
2. Bowers, W. F., and Schear, E. W.: Inguinal hernia problem viewed from standpoint of recurrent cases. Mil. Surgeon *114:* 163 (March) 1954.
3. Burton, C. C.: Inguinal canal; trihedral space, adaptation of its anatomic boundaries to modern hernia repairs, Surgery *36:* 106 (July) 1954.
4. Cooper, A. P.: The Anatomy and Surgical Treatment of Abdominal Hernia, London, C. A. Key, 1827.
5. Estes, W. L., Jr.: Five years end results in treatment of inguinal hernia, Am. Surgeon *2:* 948 (Oct.) 1956.
6. Fallis, L. S.: Direct inguinal hernia, Ann. Surg. *107:* 572 (April) 1938.
7. Harkins, H. N.: Repair of groin hernias; progress in last decade, S. Clin. North America *29:* 1457 (Oct.) 1949.
8. Harkins, H. N. and Swenson, S. A., Jr.: Cooper's ligament herniotomy; clinical experience in 322 consecutive cases, S. Clin. North America *23:* 1279 (Oct.) 1943.
9. Hoguet, J. P.: Direct inguinal hernia, Ann. Surg. *72:* 671 (Dec.) 1920.
10. Lotheissen, G.: Radicaloperation der Schenkelhernien, Centralbl. f. Chir. *28:* 548, 1898.
11. Mayo, C. W., and Keeley, J. K.: Repair of inguinal hernia with external oblique fascia, critical evaluation of results, Surg., Gynec. & Obst. *188:* 603 (May) 1949.
12. McVay, C. B., and Anson, B. J.: Inguinal and femoral hernioplasty, Surg., Gynec. & Obst. *88:* 473 (April) 1949.
13. Rienhoff, W. F., Jr.: Use of rectus fascia for closure of lower or critical angle of wound in repair of inguinal hernia, Surgery *8:* 326 (Aug.) 1940.
14. Testut, L., and Jacob, B.: Traite D'Anatomie Topographie, 3rd ed., *2:* 44 Paris, 1914.
15. Zimmerman, L. M.: Critique of McVay inguinal hernia, Surg., Gynec. & Obst. *87:* 621 (Nov.) 1948.

EDITORIAL

THE FOUR DIMENSIONS OF SURGERY

The most familiar and important dimension of surgery during the first half of the Twentieth Century has been its increasing effectiveness in the hands of highly trained and dedicated men working with increasingly better facilities and more effective team mates. During the 25 years of its existence this Journal has been devoted to the highest development of what Haggard eulogized as "The Queen of the ARTS". Surgery's effectiveness in correcting or compensating for congenital defects and repairing the damage caused by degenerative disease is attested by the fact that this issue, and indeed every issue for several years, contains more contributions to this "growing edge of knowledge" than the older and still important areas of repairing the results of trauma and combating infection.

Training young men who can carry on this tradition is and should be a major preoccupation of the leading surgical clinics. Many thoughtful people have high hopes that surgery will be increasingly effective in these areas and in making good use of the knowledge gained in basic sciences during the last two or three decades in the treatment of patients. But there are three other dimensions of surgery which seem highly important to one long deeply interested, but not directly involved, in the field.

A second dimension of surgery highly important to the general welfare is the appropriate training of young doctors who will, in fact, perform most of the "routine" operations in this country during the remainder of this century.

It has been said that in this country around 1910 it became likely that a random patient with a random complaint, who consulted a doctor chosen at random, would receive more benefit than harm from the experience. Much time and effort has been expended in upgrading the training of young surgeons certified by the American Boards as being generally competent in their field. The fact remains, however, that a very large per cent of the total surgery done in this country is done by doctors who received all their surgical training in medical school and a one-year rotating internship.

The likelihood that for several decades to come a large amount of surgery will be done by doctors whose training is relatively brief is made clear by the fact that a very large number of the total beds in the general hospitals throughout the country are located in hospitals of less than 100 beds, and that the hospital plans for most States envision the construction of many more hospitals of approximately this size, each with operating rooms which would have seemed palatial a decade or so ago. One of the major challenges of surgery at this time is seeing that in this dimension it serves society as well as possible by devising and supervising appropriate training for the young doctors who will do this large volume of surgical work.

The third dimension of surgery, highly important to each of the so-called surgi-

cal specialties as well as to surgery in general, is training in the fundamentals of surgery for the young doctors who will then seek training in each special field. Each of these special fields had its origin in the interest and skill of a few general surgeons. By concentration and effort great advances have been made in each of the fields. But we are now well into the training of the fifth generation of doctors in most of these special fields. For a while the broad background and interest to the great pioneers was partly lost sight of as the training of young doctors in these fields was increasingly devoted to technics and details and expanded in time. The weaknesses of too early and too complete specialization have now been pretty broadly recognized and most of the leaders in each of these specialties strongly advocate a thorough grounding in surgical principles. Just how this is to be accomplished, however, is not quite clear. A program that required that the young doctor first be thoroughly trained in general surgery to the level required by the American Board and then trained in his specialty would undoubtedly produce a well-trained man. There is little reason to think that a sufficient number of young men will be willing to prolong their period of training to this extent. Another challenge to surgery is to see that in this dimension it continues its fine progress by providing a way for the intending specialist to be thoroughly grounded in general surgery and its principles in a short enough time so that this and his training in his special field can be accomplished in a few years.

Just as the intellectually comfortable word of Newtonian physics and Euclidean geometry has been enriched but made more complex by the advance of mathematics, yet a fourth dimension has been added to surgery which seems likely to grow more rather than less important. In the early part of this century so much of the total welfare of the surgical patient depended upon the knowledge, skill and judgment of the surgeon that little intelligent teamwork was required. This period followed shortly after the Victorian Age with its high regard for masculine authority and most of our great surgical clinics were patterned after Germanic models. It is not surprising that the "Terror of the Operating Room and Tyrant of the Wards" was commonly the most effective surgeon and could get the modest degree of teamwork needed. Many of the triumphs of surgery within recent years has been related to far better preoperative and postoperative care of the patient and to the collaboration of skillful colleagues before, during and after the procedure. Also the national trend toward shorter working hours has heavily invaded the modern hospital. For instance, it now takes five individuals to provide coverage of one nursing position 24 hours a day seven days a week, so the team involved in caring for the patient grows larger. It is still necessary for the surgeon to carry the final responsibility for the patient's welfare and for the crucial decisions. But to discharge this responsibility and see that these decisions are carried out, the surgeon of today and tomorrow must have skill in leading an intelligent team. Whether this skill is entirely dependent upon characteristics already developed in the student before he enters medical school, or can be markedly influenced by training or by intellectual consideration of some of the things that are known about the relations of one individual to another is perhaps debatable. However, it seems reasonable to think that the lifetime

accomplishments of the young surgeon finishing his training this year will be strongly influenced by his skill in this regard as well as (by) others.

For surgery to finish as it began the Twentieth Century, at the forefront of these activities which benefit mankind, it seems likely that it will evolve training programs fully developed in all four of these dimensions. These programs will probably begin in medical school by imparting a considerable grasp of the fundamentals of surgery and the relevance of knowledge from the basic sciences to the medical student and with the selection of individuals who have or show promise of developing some skill in leading a complex team of intelligent co-workers. The program will then probably provide a phase for basic experience, training and responsibility that will give the intending specialist a firm foundation for his later training, prepare men who will practice in small communities to safely carry out a considerable range of common procedures and scrupulously avoid those procedures that require more teamwork and training than are at hand. This program must also serve as a suitable prelude to the further training of the practitioner of referred surgery and of the future investigators and teachers in the field.

ROBERT C. BERSON, M.D.
Vice-President for Health Affairs
(Of the Medical) Center and Dean of
The Medical College of Alabama
Birmingham, Alabama